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ARCHIVES OF OTOLARYNGOLOGY

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OTITIC SINUS THROMBOSIS

J. M. SUTHERLAND, M.D.

DETROIT

"Through every rift of discovery some seeming anomaly drops out of the darkness."—From Chapin's "Living Words."

Were the anomalies so frequently encountered in the anatomic structure of the area of the lateral sinus but "seeming" anomalies and were the symptoms and pathologic conditions resulting from infection of the temporal bone less variable, the old diversity of opinion as to the character and treatment of thrombosis in this area would not still persist.

Hunter,[1] in the eighteenth century, philosophically accepted nature's deviations from rule, type or form with the assurance that she still retains her fundamental regularity: "Nature is always uniform in her operations, and, when she deviates, is still regular in her deviations."

This version of the law of compensation obviously explains why when one lateral sinus, for example, is absent or greatly reduced in size the other sinus is found on exposure to be proportionately enlarged. Compensatory circulation through the dural sinuses, however, makes it often impossible to determine preoperatively the location of thrombosis in this region.

Individual cases of anatomic abnormalities are not the major problem of the otologist. He works in a unique field, in which morphologic and embryologic distinctions, together with the varying stages of childhood development, must be considered—a field which has no exact counterpart in the rest of the body.

ANATOMIC PECULIARITIES

The sixteen major dural sinuses are venous channels which drain the blood from the brain; they are devoid of valves and are situated between two layers of the dura and lined by endothelium continuous with that

Candidate's thesis for admittance into the American Laryngological, Rhinological and Otological Society.

1. Hunter, J., cited by Creighton, Charles, in Encyclopedia Britannica, ed. 10, London, Encyclopedia Britannica Company, Ltd., 1907, vol. 18, p. 367.

which lines the veins (Gray [2]). The sinuses forming a network separating the ear from the cerebral cavity are composed of the centrally situated sinus cavernosus, the sinus transversus and the sinus sigmoideus, which fuse with the peripheral veins, the sinus petrosus inferior, the sinus petrosus superior and the sinus petrosquamosus (present in children but usually absent in adults).

The part where the sinus sigmoideus fuses with the vena jugularis likewise shows topical relations to the ear. The bulbus venae jugularis, which is accommodated in the fossa jugularis, lies under the fundus of the tympanic cavity. Immediately below the bulbus, medium-sized veins empty into the vena jugularis and form a direct communication between the veins of the vertebral canal and the vena jugularis interna. However, the vena facialis communis is the only one of those inosculating somewhat more centrally into the vena jugularis interna which is of any interest to the otologist (Alexander [3]).

MORPHOLOGIC AND EMBRYOLOGIC DISTINCTIONS

In surgical operations on the temporal bone involved by endocranial complications, the lateral sinus, owing to its relatively larger size and its genetic association with the petrosquamosal sinus and the mastoid emissary vein, is of especial surgical importance. As described by Poirier [4] under the heading "The Petrosquamosal Sinus," the lateral sinus is formed by the union of two parts which are completely distinct, morphologically and embryologically—the horizontal limb and the vertical, or sigmoid, portion. The mammalia generally, with the exception of certain species of apes, have no internal jugular vein or have only a rudimentary one, which, arising as an ascending collateral branch of the external jugular vein, barely reaches the base of the skull and receives little or no blood from the organs within the skull. The lateral sinus has no vertical portion; the horizontal portion instead of bending downward continues its course, passes over the outer angle of the crest of the petrous pyramid, follows the petrosquamosal suture in a bony groove, partly converted into a canal, and leaves the skull by the temporal foramen, a short canal passing through the squamous bone. The temporal foramen opens externally above the glaserian fissure between the external auditory meatus and the temporomaxillary joint, and at this level the lateral sinus is directly continued into the external jugular vein, of which it becomes the origin, just as in man it becomes the internal jugular vein at the foramen lacerum posterius.

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2. Gray, Henry: Anatomy of the Human Body, ed. 21, Philadelphia, Lea & Febiger, 1924, p. 658.

3. Alexander, Gustav: Diseases of the Ear in Infancy and Childhood, ed. 2, Philadelphia, J. B. Lippincott Company, 1917, p. 301.

4. Poirier, cited by Ballance and Green,[7] p. 83.

This arrangement obtains in the human embryo, but at an early stage the internal jugular vein penetrates into the interior of the skull and joins the lateral sinus. The anterior portion of the petrosquamosal sinus undergoes atrophy; the blood ceases to flow in it and descends along the petrous portion of the internal jugular vein, now become the sigmoid sinus. The temporal foramen exists in the majority of adult skulls but, apart from exceptional cases, will only admit a hog's bristle. The petrosquamosal groove is met with as an anomaly in the form of a groove 3 or 4 mm. wide, following or lying alongside the suture; occasionally, instead of passing over the superior border of the petrous pyramid it tunnels through it. As for the petrosquamosal sinus (persistence of the primitive anterior segment of the lateral sinus), it can be found in 50 per cent of all specimens. Of forty-four specimens examined by Knott,[5] in eighteen it was absent on both sides, in seven it was present on both sides and in nineteen it was present on one side only.

ANATOMIC IMPORTANCE OF THE PETROSQUAMOSAL SINUS

Cheatle is cited by Politzer,[6] Ballance and Green [7] and Amberg [8] as having called attention to the importance of the petrosquamosal sinus and its significance in pathologic conditions of the middle ear. In Politzer's textbook this sinus is described as being inconstant and variable in its course. It lies in a sulcus in the middle cranial fossa, empties into the sigmoid sinus and often arrives at the external surface of the skull through a canal of the temporal bone and the zygomatic process. This sinus, when present, brings about the anastomosis between the veins of the middle ear and those of the meninges, which explains the extension of a purulent infection of the middle ear without macroscopic changes in the osseous walls of the temporal bone.

SURGICAL IMPORTANCE OF THE MASTOID EMISSARY VEIN

The surgical importance of the mastoid emissary vein has been stressed by Cheatle [9] and more recently by Hybasek [10] and Ziegelman.[11]

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5. Knott, cited by Ballance and Green.[7]

6. Politzer, A.: Diseases of the Ear, ed. 5, Philadelphia, Lea & Febiger, 1909, p. 650.

7. Ballance, Charles A., and Green, C. D.: Essays on the Surgery of the Temporal Bone, New York, Macmillan & Co., 1919, vol. 1, p. 82.

8. Amberg, Emil: Review of Lateral Sinus Thrombosis, J. Ophth., Otol. & Laryng. **23**:455 (June) 1917.

9. Cheatle, Arthur J.: Mastoid Emissary Vein and Its Surgical Importance, J. Laryng. & Otol. **40**:633 (Oct.) 1925.

10. Hybasek, J., cited by Kopetzky, S. J.: Acute and Chronic Otitis Media and Sinus Thrombosis, Arch. Otolaryng. **14**:489 (Oct.) 1931.

11. Ziegelman, Edward F.: Surgical Importance of the Mastoid Vein in Infected Lateral Sinus Thrombosis, Arch. Otolaryng. **18**:298 (Sept.) 1933.

Cheatle discussed the anatomic characteristics of this vein, its variability and its vestigial nature. This "vestigial relic" of a primitive jugular vein he found to be present in the greater number of specimens examined, and, owing to its atavism, it is subject to variation in every particular. The diameter of the canal varied from a mere thread to nearly ½ inch (1.25 cm.), with collateral changes in the size of the external jugular vein. The canal may originate above the bend of the lateral sinus groove, much lower down or in the groove itself, or it may form the lower part of the sigmoid portion of the lateral sinus. Ziegelman found the mastoid vein present in 75 per cent of the specimens he studied. He also noted its variable point of entrance into the venous circulation and its variations in size and direction. It may be invaded in infected thrombosis of the lateral sinus either by continuity of tissue or by extension of the thrombus directly. He expressed the belief that it accounted for some of the high mortality in infected sinus thrombosis and that when present it should be considered from a surgical point of view in all cases of thrombophlebitis of the lateral sinus.

ATAVISTIC ABNORMALITY OF THE MASTOID EMISSARY VEIN

The atavistic abnormality in which the mastoid emissary vein takes the place of the lower part of the sigmoid sinus was found in two instances by Cheatle [12] among fifteen hundred skulls examined in the museum of the Royal College of Surgeons. This condition was found on both sides in one skull and on the left side in the other (see plate). Laidlaw's [13] description of an Egyptian temporal bone in the museum of Cambridge University noted this atavism in the partial absence of the lateral sinus groove associated with the presence of a large mastoid foramen. The presence of an enlarged mastoid foramen in these anomalous conditions was explained by Piersol:[14]

> The horizontal portion of the left lateral sinus has been observed to be lacking or reduced to an exceedingly fine channel, and one or both of the sinuses have been observed to pass through a greatly enlarged mastoid foramen to open into the posterior auricular vein, the lower part of the sigmoid sinus being absent or being represented by a very small channel.

In some animals the mastoid emissary vein has a share in emptying the lateral sinus. In searching for the reason for this abnormality in man, Cheatle [12] quoted Wiedersheim's "The Structure of Man:" "The venous system shows unmistakable traces of a very primitive condition inherited from lower vertebrates." He also quoted Cunningham: "Abnormalities are generally due to the retention of conditions which normally are only transitory in ontogenetic development, or to

12. Cheatle,[9] p. 635.

13. Laidlaw, P. P.: Some Peculiar Features in a Temporal Bone, J. Anat. & Physiol. **37**:364, 1903.

14. Piersol, cited by Cheatle,[9] p. 636.

the acquirement of conditions which, though not as a rule present at any time in man, occur normally in some animals." Cheatle expressed the belief that the possibility of encountering atavistic abnormalities of the mastoid vein in its association with the sigmoid portion of the lateral sinus should be given more consideration by otologists.

THE SURGICAL IMPORTANCE OF VARIATIONS IN THE VENOUS SINUSES

LeDouble [15] stated that to "describe one by one all the variations in the venous sinuses is impossible, and if it were done today, it would be incomplete tomorrow." Woodhall [16] admitted that this may be true anatomically but held that the variations of surgical interest, especially in the region of the torcular herophili, may be definitely ascertained. Just why the right lateral sinus should develop to carry the greater volume of blood in the majority of persons is still a moot point, he found, and he did not accept the explanation of various authors that this primary variation is part of a phylogenetic development. It may be due to differences of outflow or to intracranial asymmetry that has been influenced in turn by asymmetrical growth of the extremity.

THE TYPES OF VARIATION OF THE VENOUS SINUSES

The five general types of variation of the sinuses in the region of the torcular are classified [16] as (1) the common pool type, classic and textbook picture of conformation, but actually encountered far less often than are the other patterns; (2) the plexiform type, which would be expected from the bilateral embryologic development of this region; (3) the ipsilateral type, in which the longitudinal sinus runs to one side; (4) the unilateral type, extremely rare, in which both the longitudinal and the straight sinus empty into one lateral sinus, the opposite sinus being attenuated or absent, and (5) the occipital type, directly concerned with the occipital sinus when it is of sufficient caliber to be of value in carrying blood. (The most common variation in the adult is the persistence of a large single or paired occipital sinus, with large marginal sinuses comparable to the pattern so common in the fetus or during the first few years of life.)

ANOMALIES OF THE LATERAL SINUS

In disagreement with the prevailing opinion that most consideration should be given to the right lateral sinus in suspected sinus thrombosis, Woodhall listed his findings in one hundred subjects examined at autopsy as: (*a*) right-sided predominance, 39 per cent; (*b*) left-sided

15. LeDouble, cited by Woodhall.[16]

16. Woodhall, Barnes: Variations of the Cranial Venous Sinuses in the Region of the Torcular Herophili, Arch. Surg. **33**:297 (Aug.) 1936.

predominance, 13 per cent; (*c*) major disproportion between the lateral sinuses, 24 per cent; (*d*) inadequate cross-circulation at the torcular, 10 to 15 per cent; (*e*) gross occipital abnormality, 4 to 6 per cent; (*f*) absence of one lateral sinus, 4 per cent and (*g*) direct relationship between the volume of the sinuses and the markings of the skull.

Eagleton,[17] in considering the variations in size and location of the lateral sinus, quoted Knott, MacEwen, Körner, Streit, Panse and Lieutaud as having recorded cases of complete or nearly complete absence of the lateral sinuses. Brown[18] reported a case of double sigmoid sinus from the knee to the occipital sinus. Braun[19] reported cases of double lateral sinus which he gathered from the articles written by Knott, Sperino, Streit, Hallet, Meckle, Dumont, Heine and Beyer. In two instances, abnormalities which Woodhall considered as "local migration" changes due to embryologic growth were noted. They were small outgrowths of the sinus wall, occurring at the sigmoid curve. (Such abnormalities were termed "lateral sinus cloacae" by Ruttin.)

Anomalies of the sigmoid portion of the lateral sinus were noted by Ruttin,[20] two illustrations of which are given. One shows an extremely wide sigmoid sinus which ends in a blind sac. It does not continue as the transverse sinus; a small transverse sinus and a small superior petrosal sinus terminate in the sac. This sinus reservoir has a narrow bony canal for the internal jugular vein; the jugular bulb is absent. The other picture shows the upper knee of the sigmoid sinus containing a blind pocket. In this case Ruttin found at autopsy a narrow sagittal and transvere sinus on the right side, both communicating with the confluens sinuum; the straight and occipital sinuses were large. Whether the presence of both illustrated anomalies on the left side has any pathologic or diagnostic significance Ruttin believes is a point of conjecture. A similar anomalous condition was noted by Furstenberg.[21] In this case the left sigmoid sinus contained a large blind sac and the sinus was filled with semiliquid blood.

ABNORMALITIES OF THE JUGULAR FORAMEN AND FOSSA

Abnormalities of the jugular foramen are frequently encountered. Linser, Kasloff, Dumont, Bairkow and Biddle (Eagleton[17]) have described great contractions of the jugular foramen. They observed

17. Eagleton, W. P.: Circulatory Disturbances Following Ligation of the Internal Jugular Vein, Arch. Otol. **35**:91, 1906.

18. Brown, J. M.: A Double Sigmoid Portion of the Lateral Sinus, Tr. Am. Laryng., Rhin. & Otol. Soc. **27**:302, 1921.

19. Braun, Alfred: Sinus Thrombophlebitis, New York, Paul B. Hoeber, Inc., 1928, p. 55.

20. Ruttin, Erich: Kloaken- und Blindsackbildung am Sinus sigmoideus, Aus der Universitätsklinik für Ohren-, Nasen- und Kehlkopfkrankheiten in Wien. Vorstand des Ohrenambulatoriums am Rudolfspital in Wien.

21. Furstenburg, A. C.: Personal interview with the author.

variations in its diameter from that of a bristle to that of a probe, while Zuckerkandl described a specimen in which the right jugular vein was eight times the size of the left. Obviously, the vein can be no larger than the foramen through which it passes. The size of the jugular foramen not only determines the size of the vein but influences the size of the jugular fossa. The jugular fossa [22] is of variable size and depth and undergoes cribriform perforations or complete detachment. Politzer [23] noted a number of specimens in which the jugular fossa extended up to the internal auditory meatus and showed dehiscences.

CHARACTER OF THROMBI

The old controversy as to the character of a thrombus and the resulting diversity of opinion as to its treatment in otitic sinus thrombosis revolves about the question as to whether a thrombus may be a protective measure by nature to prevent micro-organisms infecting the middle ear from entering the general circulation, or whether, as the minority of otologists believe, a thrombus in such conditions is infected from the beginning.

Welch [24] said:

> The distinction between bland thrombi and infective thrombi is an old and important one. Recent attempts to refer all thromboses, formerly called marantic, to the direct invasions of micro-organisms and to phlebitis, go beyond demonstrated facts. Thrombosis may be incited by chemico-toxic changes in the blood or by mechanical pressure of an inflammatory mass, from a focus of infection causing a bulging inward of the sinus wall, with later disturbance of the endothelium. Although bacteria found in the intima may have gained access either through the vasa vasorum or through the lymphatics, the invasion of micro-organisms may produce an extensive bacterial inflammation of the sinus wall, even with bulging of the intima into the lumen of the vessel, without producing thrombosis; a lesion of the endothelium of the sinus wall must be present before thrombosis takes place. The thrombus at first is a mural one, but by continued growth it fills the vessel becoming an occluding thrombus.

A thrombus is in effect essentially a protective mechanism. Its formation may prevent the access of bacteria into the general circulation. It is the secondary infection of the clot and not the occurrence of the thrombus that constitutes the danger, or the condition characterized by its presence. In some rare cases [25] in which septicemia or pyemia occurs from severe disease of the temporal bone without obvious infection or obstruction of the lateral sinus and jugular vein, the infection has been so virulent that the protective effects of thrombosis have not been realized.

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22. Gray,[2] p. 142.
23. Politzer,[6] p. 22.
24. Welch, William H.: Thrombosis, in Papers and Addresses, Baltimore, Johns Hopkins Press, 1920, vol. 1, p. 126.
25. Ballance and Green,[7] p. 123.

ETIOLOGY OF THROMBOSIS

The three causes [26] of thrombosis are: (1) alterations in the blood, (2) mechanical disturbances of the circulation and (3) lesions of the vascular wall, especially the endothelium. The theory of dyscrasia is the oldest. John Hunter introduced and Cruveilhier elaborated the conception of primary phlebitis with consecutive plugging of the vein; Baillie, Laennec, Davey and others emphasized stasis as a cause of intravascular clotting. In 1846 Virchow, a proponent of the mechanical explanation of thrombosis, stated that the thrombus is the primary and essential phenomenon and inflammation of the wall of the vessel, if present, merely a secondary effect. The experiments of Brücke showed the importance of the integrity of the vascular wall and led to the general recognition that the influence of the endothelial lining of the vascular channel in keeping the blood fluid is of the first importance. This influence appears to be partly physical and partly chemical. While there is little definite information about chemical activity of the normal endothelium, the theory that necrotic endothelial and intimal cells may liberate thromboplastic substances is in accordance with both physiologic and pathologic observations, and thrombosis may thus be incited through chemical as well as physical influences.

STRUCTURE OF THROMBI

According to Welch,[27] there are two main anatomic groups of thrombi, red and white; the elements which enter into the composition of fresh thrombi are blood platelets, fibrin, leukocytes and red corpuscles. These elements are present in varying number, proportion and arrangement; hence the great diversity in appearance and structure of different thrombi. Zahn, in 1872, was the first to make a systematic experimental study, mainly in frogs, of the mode of formation of thrombi. His conclusions were that the process was initiated by the accumulation of white corpuscles. They disintegrated, giving rise to granular detritus and fibrin. Mantegazza held this view in 1869, and it was confirmed by Pitres in 1876.

Continued experimental studies, especially on mammals, led to the opposition of Zahn's conclusions and favored the opinion, now generally accepted, that the white thrombus starts as an accumulation of blood platelets. The investigators chiefly concerned in the establishment of this doctrine were Osler, Hayem, Bizzozero, Lubnitzky, Eberth and Schimmelbusch.

Welch, in 1887, verified by a microscopic study at intervals of minutes, the development of thrombi. The material composing the youngest thrombi formed from the circulating blood appeared as a soft,

26. Welch,[24] p. 121.
27. Welch,[24] pp. 110-112.

viscid homogeneous gray translucent substance. Microscopically it was made up chiefly of platelets, which were seen as pale round irregular bodies about one-quarter the diameter of a red blood cell. Leukocytes rapidly increase in number and within the first fifteen to thirty minutes may be considered the essential constituent of the thrombus. A thrombus is at first soft and moist, but by extraction and extrusion of fluid it becomes more compact, firmer, drier and more granular.

Such a dried and compact thrombus may be encountered in the internal jugular vein after thrombosis of the lateral sinus when previous ligation has been performed.

GROWTH, METAMORPHOSES AND ORGANIZATION OF THROMBI

Welch [28] stated that a primitive, or antochthonous, thrombus caused by local conditions may be the starting point of a continued or propagated thrombus extending along the course of the vessel and perhaps into communicating vessels. A continued thrombus is also often spoken of as secondary. A venous thrombus not only extends in the direction of the blood current, but at times extends in the opposite direction, causing retrograde thrombosis.

Of special importance are the liquefactive softenings which may occur in thrombi. These are distinguished as bland, septic or putrid softenings. The septic, or purulent, softening met with most frequently in infective thrombophlebitis is a true suppuration, being the result of accumulation of polymorphonuclear leukocytes with fermentative liquefaction of the thrombus. Pyogenic bacteria, most frequently streptococci, are present in the thrombus and in the walls of the vessel. Putrid softening is due to invasion by putrefactive bacteria, which produce a dirty brown or green clot of a foul odor. These softenings often lead to separation of thrombotic fragments to be transported by the circulation as emboli—bland, septic or putrid, according to the nature of the process.

Welch said:

> One of the most interesting adaptive pathological processes is the so-called organization of thrombi, which is the substitution for the thrombus of vascularized connective tissue. The thrombus itself takes no active part in the process, but behaves as a foreign body. It is gradually disintegrated and absorbed, largely through the activities of phagocytes. The new tissue springs from the wall of the vessel, the tissue-forming cells being derived both from the endothelium and from other fixed cells in the wall. New vessels spring from the vasa vasorum. Lacunar spaces in the thrombus, or between the thrombus and the vascular wall, may become lined with endothelium, and also serve as channels for the circulating blood. These new vessels may establish communication with the lumen of the thrombosed vessel above or below the thrombus, or on both sides. The new tissue, which at first is rich in cells, becomes fibrous, and contracts. The result

28. Welch,[24] pp. 117-121.

may be a solid fibrous plug, or a cavernous structure with blood-spaces; or, by disappearance of the septa, a restoration of the lumen, with perhaps a few fibrous threads or bands stretching across it, as in the normal cerebral venous sinuses.

There are great diversities in individual cases as to the rapidity of onset and the course of the organizing process. These differences depend on the location of the thrombus, the condition of the wall of the vessel, the general state of the patient and the presence or absence of infection. The sinus may be so diseased as to be incapable of furnishing any new tissue. The presence of pyogenic bacteria prevents or delays the process of organization. The process is a proliferative angiitis which leads to the closure of a vessel after ligation. If the ligature is applied aseptically and without injury to the internal coats of the vessel, usually no thrombus is formed or only a small one. The formation of the thrombus is of no assistance in securing obliteration of the ligated vessel; in fact, it impedes the development of the obliterating process.

PHYSIOLOGIC PECULIARITIES INFLUENCING THE FORMATION OF VENOUS THROMBI

Venous thrombi, according to Welch,[29] may result from both local and general causes. The local causes are: phlebitis, traumatism, compression, inflammation or other lesions of the surrounding tissues, and connections of venous terminals with septic or gangrenous foci.

The physiologic peculiarities, partly local and partly general, which come especially into consideration are: the importance of slowing and other irregularities of the circulation, very low or negative blood pressure in the cerebral sinuses, fixation of the venous wall to the bone, absence of valves, damage to the venous wall from impoverished and insufficient blood supply or prolonged contact with micro-organisms and toxic substances, and the composition of the venous blood, particularly the higher content of carbon dioxide in the blood. Trabeculations which cross these sinuses may be a factor in retarding the blood current. Von Recklinghausen attached much more importance to the whirling, or eddying, motion than to mere slowness of the circulation. He pointed out that eddies are produced when the blood enters normally or pathologically dilated channels from smaller ones or passes into a cul-de-sac or over obstructions. All these physiologic factors have their influence on the production of thrombi.

Lancereaux,[30] in 1862, enunciated the following law as an explanation of the common site of thrombi in the cerebral sinuses:

> Marantic thromboses are always formed at the level of the points where the blood has the greatest tendency to stasis, that is, at the limit of the action of the forces of cardiac propulsion and of thoracic aspiration.

29. Welch,[24] p. 136.
30. Lancereaux, cited by Welch,[24] p. 137.

Welch [31] said:

> There are serious physiological objections to the physical conceptions of the circulation underlying this so-called law, which in any event cannot be accepted in the exclusive form given to it by Lancereaux. As the collective sectional area of the veins steadily diminishes from the capillaries to the heart, the average speed of the blood must be greater in the large veins than in the small ones, if the circulation is to continue for any length of time; and this remains true even when the energy of the blood current is feeble.

The anatomic structure and location of the sigmoid sinus and jugular bulb predispose these structures to thrombosis from disease of the temporal bone. The existence of wide, or ampulla, dilatations, sometimes encountered in the cerebral sinuses, especially in the lateral sinus and the jugular bulb, may induce a whirling, or eddying, motion or other irregularities of the circulation and may be a further factor in causing thrombosis in this region.

PATHOLOGIC CONSIDERATIONS

Every purulent infection of the middle ear carries with it the possibility of a number of serious intracranial complications. In infancy and early childhood the blood vessels and lymphatics of the mucosa of the middle ear are in more direct connection with the mastoid, the jugular bulb and the meninges than in adult life. Therefore, in a case of tympano-antral suppuration, extension of an infection to these structures frequently results in some endocranial lesion, such as meningitis, subdural abscess or primary phlebitis of the jugular bulb or thrombosis, without macroscopic evidence that the osseous structure of the mastoid is involved.

Disease of the temporal bone may be secondary to infection of the tympano-antral cavity or may occur independently of it. Infection of the temporal bone may give rise to: [32] (1) more or less extensive local destruction, (2) extension of infection to surrounding tissues, (3) transference of infective particles to and formation of fresh foci in other parts of the body or (4) general systemic intoxication or infection. Whether earlier operation would reduce the mortality from endocranial complications remains conjectural. Yet at the operation one is often surprised at the extent of the destruction of bone in spite of every laboratory aid for diagnosis and although the operator has lived up to every indication for operation.

RELATIVE IMPORTANCE OF THE DURAL SINUSES

In a consideration of the relative importance of the dural sinuses in endocranial infection of otitic origin, the larger size of the lateral sinus,

31. Welch,[24] p. 137.
32. Ballance and Green,[7] p. 113.

the confluence of its sigmoid portion with the internal jugular vein and bulb and the close topical relation to the diseased temporal bone center attention on this sinus in locating suspected thrombosis. However, Ballance and Green [33] said:

> Too much stress has been laid on the lateral sinus and jugular bulb as though they alone were subject to infective thrombosis. The size of the lateral sinus concentrates the mind too exclusively upon it, while in pyaemia resulting from disease in other regions of the body, no large vein being in near proximity to the disease, attention is not distracted from the numerous small veins, each one of which may harbour an infective thrombus which may equally produce the disease.
>
> The process of disease in the temporal bone and in the tissues around it is one continuous infective process. It is a mistake to think of the infection of the osseous, dural, and blood tissues as independent pathological conditions. The differences are anatomical and physiological, not pathological. As the spread of the infection is by continuity of tissue, the infection of the dura, for obvious anatomical reasons, precedes that of the blood. The march of infection will thus be in this order: parasinusitis, sinusitis, blood infection. Infection of the blood suddenly brings into being a new factor, for it is infection of a tissue of liquid texture, which has the physiological property of constantly travelling through all the tissues of the body.

Other sinuses may be involved from disease of the temporal bone. The superior sagittal sinus [34] may be involved, particularly in infancy and childhood, from extensive retrograde thrombosis to the torcular Herophili or through the anastomotic veins of Labbé and Trolard and adjacent cerebral veins. Primary thrombi [35] may occur in the superior petrosal sinus, especially from suppuration of the labyrinth or the petrous pyramid, and also in acute suppuration in the tympanum in scarlet fever. The inferior petrosal sinus may be involved from the petrous pyramid, the jugular bulb or the cavernous sinus. Passow [36] and others have reported cases of fatal pyemia in which the inferior petrosal sinus contained a septic thrombus discovered at autopsy. Paulson,[37] Brown and Bowman [38] and others have reported cases of thrombosis of the inferior and superior petrosal sinuses (autopsy reports). Primary thrombosis of the jugular bulb may occur in chil-

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33. Ballance and Green,[7] pp. 122-123.

34. Doyle, John B.: Obstruction of the Longitudinal Sinus, Arch. Neurol. & Psychiat. **18**:375 (Sept.) 1927.

35. Brieger, cited by Ballance and Green,[7] p. 100.

36. Passow, cited by Ballance and Green,[7] vol. 2, p. 492.

37. Paulson, cited by Ballance and Green,[7] vol. 2, p. 493.

38. Brown, J. M., and Bowman, Robert J.: A Case Report of Thrombosis of the Lateral Sinus, Inferior Petrosal Sinus and Opposite Lateral Sinus: Autopsy Finding, Laryngoscope **43**:664 (Feb.) 1933.

dren. Fremel,[39] Schlander,[40] Maybaum and Goldman [41] and Friesner [42] reported such findings, while Dintenfass [43] reported a case in an adult with normal mastoid findings at operation. Fremel expressed the belief that an important mode of pathogenesis of primary thrombosis of the jugular bulb is direct contact with the infection in the peribulbar cells of the mastoid process, called "threshold cells" by Bayer, while the opinions of other otologists seem to favor the infection of the bulb from the direct extension of the infection from the tympanic cavity into the dome of the bulb.

The virulence of the pathogenic organism and the inherent resistance of the host are of obvious importance. Friesner [44] described an invasion into the general circulation as streptococcemia, staphylococcemia and pneumococcemia, each of which expresses a clearcut idea. Streptococcus haemolyticus, having a particular liking for the sigmoid sinus, is most often the organism producing thrombosis, while the type III pneumococcus or Streptococcus mucosus-capsulatus more often produces widespread phlebitis through the smaller veins and invariably affects the endocranium after invading the mastoid. Kopetzky [45] stated that the distribution of metastatic lesions depends on the type of organism present and its inherent selectivity.

SYMPTOMATOLOGY AND DIAGNOSIS

Coates and his associates [46] stated: "The classic symptoms of lateral sinus thrombosis may be grouped under three heads: (1) Those due to systemic phenomena; (2) Cerebral symptoms, and (3) Septic." All these present highly distinctive characteristics, which, except in the atypical forms, may render the diagnosis possible at an early stage of the disease, provided a reliable history is available.

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39. Fremel, F., cited by Kopetzky, S. J.: Acute and Chronic Otitis Media, Sinus Thrombosis and Petrous Apex Suppurations, Arch. Otolaryng. **18**:344 (Sept.) 1933.

40. Schlander, E., cited by Kopetzky, S. J.: Acute and Chronic Otitis Media, Sinus Thrombosis and Petrous Apex Suppurations, Arch. Otolaryng. **18**:344 (Sept.) 1933.

41. Maybaum, J. L., and Goldman, J. J.: Primary Jugular Bulb Thrombosis, Arch. Otolaryng. **17**:70 (Jan.) 1933.

42. Friesner, Isidore, and Druss, J. D.: Intracranial Complications of Otitic Origin, Arch. Otolaryng. **15**:356 (March) 1932.

43. Dintenfass, H.: Primary Jugular Bulb Thrombosis in an Adult, Arch. Otolaryng. **20**:422 (Sept.) 1934.

44. Friesner, Isidore: Infections of the Middle Ear, Arch. Otolaryng. **14**:257 (Sept.) 1931.

45. Kopetzky, S. J.: Acute and Chronic Suppuration Otitis Media, Including Acute Infection of the Blood Stream, Arch. Otolaryng. **2**:379 (Oct.) 1925.

46. Coates, George M.; Ersner, M. S., and Persky, A. H.: Lateral Sinus Thrombosis with a Review of the Literature, Ann. Otol., Rhin. & Laryng. **43**:419 (June) 1934.

In describing the differential diagnosis of sinus thrombosis from abscess of the brain and meningitis, Cahill [47] said:

> We are aided by the fact that each typical case of these three intracranial lesions of middle ear suppuration has distinctions clear and evident. By its general appearance, by its temperature curve, by its spinal fluid picture, by its field of vision and fundi; each differs markedly from the others. The dull apathetic brain abscess patient stands out in clear contrast to the anxious restless meningitic or the bright animated (early) thrombotic.
>
> Equally as distinct are the temperature charts; the normal or subnormal curve of the abscess case, the consistently high temperature level of the meningitic, and the picket fence septic form of the infected sinus.
>
> The spinal fluid pictures are nearly as characteristic. Extension or close proximity of the toxins of the abscess or sinus lesions to the pia-arachnoid spaces may vary the form of these pictures to a puzzling extent.

ATYPICAL MANIFESTATIONS

With atypical manifestations complicated with cerebral symptoms or disturbances, such as ocular paresis or paralysis,[48] convulsions,[49] stupor, choked disks,[50] homonymous hemianopia, hemiplegia, aphasia,[51] meningeal irritation [52] or other puzzling symptoms, the otologist needs every diagnostic aid at his command to make a diagnosis. Many of these symptoms may be due to a mild infection, a cachectic or debilitating condition, a low virulence of the organism, a sterile clot,[53] back pressure or venostasis of the cortex. Coates, Ersner and Persky [46] expressed the belief that many of these baffling disturbances of otitic origin may be traceable primarily to anomalies of the lateral sinus and the jugular foramen. The lack of proper collateral circulation, especially between the lateral sinuses, Klestadt [54] stated, is a factor in producing

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47. Cahill, H. P.: Differential Diagnosis of Intracranial Lesions from Middle Ear Suppuration, graduate lecture at meeting of the American Academy of Ophthalmology and Otolaryngology, 1936.

48. Greenfield, Samuel D.: Etiology and Pathology of Paralysis of the Abducens Nerve Associated with Sinus Thrombophlebitis, Arch. Otolaryng. **19**: 336 (March) 1934.

49. Courville, Cyril B., and Nielsen, J. M.: Cerebral Manifestations Following Acute Otitis Media in Infancy and Childhood, Am. J. Dis. Child. **49**:1 (Jan.) 1935.

50. Turner, A. Logan: Diseases of the Nose, Throat and Ear, ed. 4, Baltimore, William Wood & Company, 1936, p. 385.

51. Nielsen, J. M., and Courville, Cyril B.: Intracranial Complications of Otogenous Thrombosis of the Lateral Sinus, Ann. Otol., Rhin. & Laryng. **46**:11 (March) 1937.

52. Birsner, L. J., and Wolff, Dorothy: Thrombophlebitis Following a Case of Chronic Otitis Media, Ann. Otol., Rhin. & Laryng. **39**:1045 (Dec.) 1930.

53. Holderman, J. W.: Lateral Sinus Thrombosis Without Elevation of Temperature, Arch. Otolaryng. **1**:488 (May) 1925.

54. Klestadt, Walter: Zerebrale Symptomenkomplexe bei otogener Sinusphlebitis, Ztschr. f. Laryng., Rhin., Otol. **13**:83, 1925.

some of the cerebral symptoms of back pressure. He found that an unusual situation is created, however, when after an operation for sinus thrombosis or phlebitis an intracranial symptom complex sets in which arouses suspicion of a further otogenic complication without the possibility of a definite diagnosis, and he emphasized the statement that the symptoms in four cases were such that a clinical diagnosis of abscess of the brain was made in every instance. This diagnosis was reversed after the spinal punctures at intervals gave negative results, and the patients continued to get well.

Nielsen and Courville[54a] found:

> We may find, therefore, as a result of thrombosis of the lateral sinus, a wide variety of intracranial lesions, some transitory, others of serious and often of lethal character. Among the first group may be included focal venous infarctions with meningeal and cortical edema. Among the latter are to be included subdural abscess and hemorrhage, septic meningitis, gross areas of red softening and cerebellar or cerebral abscess.

In a later article[51] they stated:

> These lesions produced by thrombosis of the lateral sinus are extremely variable and depend upon: (1) the pre-existing anatomic situation, (2) the virulence of the infecting organism, (3) the suddenness with which the channel is occluded especially if no thrombus is present, (4) the extent of thrombosis and (5) the character of the local venous lesions.

MECHANISM OF CHOKING OF THE OPTIC DISK

Turner in his investigation of and experience with thrombosis of the lateral sinus reported that choking of the optic disks occurs in two thirds of the cases. Benedict[55] said:

> With the knowledge that choked disc may occur in surgical diseases of the mastoid with or without thrombosis of the lateral sinus, the only conclusion one can reach regarding the significance of choked disc is that some intracranial involvement has taken place, but the nature of this involvement is not manifest.

Parker[56] held that there are three principal theories to account for the causation of choking of the disks: the inflammatory, the toxic or chemical and the mechanical. The mechanical theory depends on increased intracranial pressure, increased intravaginal pressure, disturbance of the return flow of blood and obstruction of the flow of lymph to the subarachnoid spaces. He found by experimentation that increased intracranial pressure in the occipitoparietal region produces choking of

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54a. Nielsen, J. M., and Courville, C. B.: Pseudo-Abscess of the Brain: Intracranial Disease During Otitis Media Simulating Encephalic Abscess, Ann. Otol., Rhin. & Laryng. **43**:972 (Dec.) 1934.

55. Benedict, W. L.: Cranial Sinus Thrombosis: Ophthalmological Aspect, Surg., Gynec. & Obst. **52**:464 (Feb.) 1931.

56. Parker, Walter R.: Relation of Choked Disk to the Tension of the Eyeball, J. A. M. A. **67**:1053 (Oct. 7) 1916.

the disks in the eye of the least tension and that 78 per cent of the clinical cases observed bear out the theory that choking of the disks in cases of increased intracranial pressure manifests itself first in the eye of lesser tension.

Cushing and Bordley [57] expressed the belief that the changes are due, first, to mechanical and, second, to toxic or inflammatory causes. De Schweinitz [58] said:

> In general terms, it is probable that choked disc is produced by a combination of factors. In this combination, increased intracranial tension or pressure is most prominent, and the mechanical theory of its pathogenesis offers the most satisfactory explanation.

REQUIREMENTS FOR SURGICAL INTERVENTION

In puzzling cases of atypical manifestations the only requirement is to recognize that some intracranial disease of otogenic origin is present, this being a sufficient indication for immediate operation. Thrombophlebitis, meningitis, subdural abscess or abscess of the brain may coexist in any combination of complications. Differential diagnosis in such conditions is only of subordinate value. The otologic surgeon [59] shares the point of view of the general surgeon; that is, it is often necessary to be content with having localized a disease sufficiently to permit the institution of surgical measures at the right place, and operative findings generally will furnish the exact diagnosis.

ADJUNCTS IN DIAGNOSIS

Blood Cultures.—Positive blood culture with symptoms of sinus thrombosis, associated with the history of suppuration of the middle ear, may or may not indicate actual thrombosis in the sigmoid sinus. Negative blood cultures do not necessarily exclude sinus thrombosis. Usually the clinical picture presented by the patient is the most valuable guide. To wait until a positive blood culture is obtained may prove fatal.

Roentgenograms.—Beck [60] said "The value of stereoscopic radiography cannot be overestimated in the studying of anatomy of the human skull, especially the mastoid process, without dissection or destruction of any part of the skull." It is also useful in showing the location, size and anomalies of the lateral sinus (Wanamaker [61]). Ersner and

57. Cushing and Bordley, cited by Parker, Walter R.: The Mechanism of Papilledema, Arch. Neurol. & Psychiat. **14**:31 (July) 1925.

58. de Schweinitz, George E.: Disease of the Eye, ed. 10, Philadelphia, W. B. Saunders Company, 1924, p. 554.

59. Alexander,[3] p. 309.

60. Beck, Joseph C.: Radiographic Study of Comparative Anatomy of the Sinuses and Mastoids of Some of the Lower Animals, Tr. Am. Acad. Ophth., 1910, p. 260.

61. Wanamaker, A. T.: Considerations and Suggestions in the Handling of Lateral Sinus Thrombosis, Laryngoscope **40**:787 (Nov.) 1930.

Myers [62] recently showed that the right lateral sinus may be absent and demonstrated irregularities in the size and shape of the jugular foramen. These findings were demonstrated preoperatively and confirmed at the operation. A large emissary vein on the right side was also traced to its entrance into the jugular bulb.

From the otologist's standpoint, the most important thing is to know whether complications exist or are in the process of development. The roentgen examination can give no information as to these points. However, roentgen examination of the temporal bone, especially the mastoid process, reveals its character and the distribution of its cellular structure —that is, whether the bone is diploic, pneumatic, sclerotic, infantile or non-pneumatized or presents a combination of these characteristics. The particular condition of the development of the mastoid process as determined by roentgen examination should predicate what conclusion may be reached relative to the spreading of an otitic infection. The extent of the cells and the areas of broken-down bone, situated close to the dura or a sinus, may, however, indicate that there is danger of perisinuous or extradural abscess.

Dynamics of the Spinal Fluid in the Diagnosis of Thrombosis of the Lateral Sinus.—The Ayer-Tobey modification of the Queckenstedt test depends on the increase of the spinal fluid pressure in the presence of occluding thrombosis when digital pressure is applied to the uninvolved vein. Its reliability is particularly marked in the presence of occluding thrombosis, but when the thrombus is only mural there is opportunity for a certain percentage of error. Coates, Ersner and Persky [46] have found this test to be of value only in addition to the interpretation of other clinical symptoms. Meltzer [63] found the test to give correct results in from 75 to 80 per cent of cases. He thinks that the test continues to be one of the best diagnostic aids in cases of thrombosis of the lateral sinus. Dowmann, Kindler and Perwitzschky stated that the test gives a definite answer as to whether a thrombus is present and on which side it is located when pressure is properly applied; yet in the clinic of the Sabbatsberg Hospital the test was found rather unreliable (Frenckner [64]).

Woodhall [16] questioned the validity of the Queckenstedt test in the diagnosis of thrombosis of the lateral sinus and grouped into three

62. Ersner, M. S., and Myers, D.: Aid to Interpretation of Intracranial Complications Resulting from Venous Circulatory Disturbances of the Temporal Bone, Offered by X-Ray of the Lateral Sinus and Jugular Foramen, Laryngoscope **43**: 800 (Oct.) 1933.

63. Meltzer, Philip A.: Treatment of Thrombosis of the Lateral Sinus, Arch. Otolaryng. **22**:131 (Aug.) 1935.

64. Frenckner, P.: Some Experiments with Venosinography: Contributions to the Diagnosis of Otogenous Sinus Thrombosis, Acta oto-laryng. **20**:477, 1934.

classes certain contradictory observations that have been noted in the original studies:

1. Those cases in which a unilateral and false negative Queckenstedt reaction has been obtained in spite of the fact that thrombosis of the lateral sinus has been demonstrated clinically. Canuyt and Klotz and Gaillard and Mayoux expressed the belief that contradictory results are obtained because of dilatation of anastomosing venous channels, particularly the inferior petrosal sinus, the thyrolinguofacial trunk, the ophthalmic vein and the pterygoid plexus. Other observations illustrating the mechanism of this collateral circulation have been reported by Whitehead, Frenckner, Stauffer, Aubin and Kopetzky.

2. This group embraces those rare cases in which a positive response to the test is obtained on both sides, although pressure on both internal jugular veins simultaneously produces an adequate response. It is probable that the unusual results are due to extensive channels of cross-circulation from one lateral sinus to the other.

3. This group comprises those instances in which a unilateral positive response to the Queckenstedt test is obtained although the lateral sinus is not thrombosed and no abnormality of the sinus can be demonstrated. This may result from a perisinuous abscess or a tumor in the cerebellopontile angle, causing complete or partial obliteration of the transverse sinus.

Venosinography.[64]—This is one of the more recent tests, and while it is too early to make deductions as to its value, the future will show how applicable it is in practice. The drawback of the trephine operation should not discourage its use, provided the test gives positive information. Venosinography is based on the same principle as arteriography; that is, a contrast material, such as thorium dioxide, is injected into the venous blood stream for the diagnostic localization of obstruction.

SUPPORTIVE TREATMENT

The supportive treatment consists of: 1. Transfusions of whole or immunized blood, given in quantities from 100 to 150 cc. at frequent intervals. 2. Serum treatment with antistreptococcus serum, given intramuscularly or intravenously, and convalescent human serum, given intravenously or beneath the fascia lata. 3. Chemicotherapeutics: sulfanilamide and its derivatives orally and intramuscularly, Pregl's solution of iodine and metaphen, given intravenously. However, these drugs may produce toxic manifestations.

SURGICAL TREATMENT

The following surgical technics have been used since the operation on the lateral sinus was first advised:

1. A simple incision of the sinus wall and removal of the thrombus.

2. Removal of the thrombus and ablation of the sinus.

3. Removal of the thrombus, ablation of the sinus and ligation of the internal jugular vein.

4. The Alexander operation, which is described as follows: Removal of the thrombus, ablation of the sinus and ligation and severance of the internal jugular vein, its cephalic end being sutured to the skin and irrigation being effected from above through the segment composed of the sigmoid sinus, the jugular bulb and the cephalic end of the jugular vein. Alexander [65] stated: "This arrangement makes provision by a single operation for everything that is necessary for the surgical treatment and healing of otitic pyemia."

5. The following operation, first performed by Grunert [66] and modified by Voss, Laval and others, was formerly popular in Europe: removal of the thrombus from the sinus, ablation of the sinus and resection of the internal jugular vein and bulb. According to many, this procedure further meets the surgical requirements of hypothetic cases.

In considering the diversity of opinion concerning the treatment of otogenous sinus thrombosis as advocated by the leading otologists, one is confronted with the classic procedure of ligation of the internal jugular vein, which was first recommended by Zaufal [67] in 1880 and which was based on the false assumption that ligation of the internal jugular vein bars the way of infectious thrombi to the heart or other parts of the body. The principle on which this procedure is based was first applied successfully in the treatment of extradural venous inflammation by John Hunter,[68] in the first half of the eighteenth century.

Lillie [69] said that clinical and surgical judgment is often challenged when suitable measures must be selected for patients with infection of the blood stream from disease of the temporal bone. He expressed the belief that much better results may be obtained, especially in sinus thrombosis, if surgical procedures are adapted to each case individually. Coates and his associates [46] Furstenberg,[70] Almour,[71] Dixon,[72] Rott [73]

65. Alexander,[3] p. 319.

66. Grunert, cited by Politzer,[6] p. 662.

67. Zaufal, cited by Bondy, Gustav: Die otitische Sinusthrombose, in Lewis, B.: Medizinisches Vademekum für Ohrenarzte, Vienna, Karl Brakl, 1921, p. 41.

68. Hunter, John: Treatise of the Blood, New York, Longmans, Green & Co., 1837, vol. 3, p. 581.

69. Lillie, H. I.: Suppuration of Temporal Bone Accompanied by Infection in the Blood Stream, Arch. Otolaryng. **18**:630 (Nov.) 1933.

70. Furstenburg, A. C.: Personal communication to the author.

71. Almour, R.: Basis for Selection of the Type of Procedure in Sinus Thrombosis, Laryngoscope **44**:454 (June) 1934.

(Footnotes continued on next page)

and others agreed with Lillie that ligation, severance or resection of the internal jugular vein should be reserved, modified or omitted according to the particular pathologic conditions which are present at the time of operation. However, Ruttin,[74] Portmann,[75] Beck,[76] Shurly,[77] Campbell [78] and others advocated the ligation of the internal jugular vein in all cases of thrombosis of the infected lateral sinus.

From the standpoint of the clinician, three things are necessary in cases of infection of the mastoid with sinus thrombosis:

1. Eradication and drainage of the primarily infected area.

2. Ligation of the internal jugular vein in an attempt to prevent metastases and infiltration of septic material from the thrombosed sinus or vein into the circulation.

3. Realization that tying off or resecting an infected thrombosed vein is not enough. What might be called the "pathologic pressure" of the process is not released unless the sinus or the vein or both if necessary are opened, evacuated and allowed to granulate instead of being left to uncertain attempts on the part of the tissues to absorb them or to wall them off.

Failure to appreciate the third indication is the one factor that has most often led to unfortunate results and thwarted the beneficial effects of attending to the first two.

Some otologists contend that unless all the venous channels are blocked pus and bacteria may be conveyed from the auricular region by other vessels aside from the internal jugular vein, such as the condyloid and vertebral veins or the marginal and basilar sinuses, or by retrograde extension through the transverse portion of the lateral sinus or through the petrosal sinuses. Others believe that ligation of the internal jugular vein is the most important factor in preventing further infection, as this vein is the principal centripetal channel conveying the contents of the infected sinus.

Batson [79] has recently shown by his series of corrosion preparations the many intercommunications in the venous system of the head

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72. Dixon, O. Jason: A Departure in the Management of Infections of the Sigmoid Sinus, Arch. Otolaryng. **2**:453 (Nov.) 1925.

73. Rott, O. M.: Why Ligate the Jugular Vein in Cases of Lateral Sinus Thrombosis? Arch. Otolaryng. **14**:272 (Sept.) 1931.

74. Ruttin, E.: Personal communication to the author.

75. Portmann, G., cited by Kopetzky, S. J.: Acute and Chronic Otitis Media: Sinus Thrombosis and Petrous Apex Suppuration, Arch. Otolaryng. **18**:344 (Sept.) 1933.

76. Beck, J. C., cited by Oppenheimer, Seymour: Discussion on Some Remarks on Lateral Sinus Thrombosis, Tr. Am. Acad. Ophth., 1910, p. 319.

77. Shurly, Burt R.: Personal interview with the author.

78. Campbell, Don M.: Lateral Sinus Thrombosis, Laryngoscope **2**:775 (Oct.) 1922.

79. Batson, Oscar V.: Personal communication to the author.

and neck. These new preparations show in a rather complete way the fact that many channels may conduct blood from the structures in the temporal bone and from the temporal bone itself to the general circulation. He has shown the connections between the lateral sinus and the veins of the brain and the numerous anastomoses of the minute veins of the temporal bone with the lateral sinus. Medially, he has demonstrated the several connections from the area of the temporal bone, the superior petrosal sinus to the basilar plexus and the inferior petrosal sinus to the basilar plexus. The rich interconnections between the basilar sinus, the cavernous sinuses and the pterygoid veins, as seen in the corrosion preparation, form a large single midline plexiform mass. The pericarotid plexus joins that midline system with the jugular bulb, as does also the sinus of Englisch. The foregoing connections, together with the several intercommunications of the pterygoid plexus, make the problem of thrombosis of the lateral sinus particularly complex.

With many of these anatomic channels inosculating into the lateral sinus and several intercommunications between the external and internal jugular veins, it is not surprising that ligation of the internal jugular vein is not always successful in preventing metastasis and that metastases do often occur in spite of the ligation. However, to discard surgical intervention on the jugular vein because it does not influence the retrograde growth of a thrombus or prevent metastases seems to be illogical. There are many anatomic and pathologic conditions which preclude the possibility of successfully treating thrombosis in certain cases, and these factors may explain why such a high mortality rate prevails even when operations have been performed in a manner that seems to fulfil all technical and surgical requirements.

SUMMARY

Anomalies in the area of the lateral sinus are often a serious factor in complicating the course of otitic sinus thrombosis. Interference with collateral circulation, especially the lack of intercommunication between the lateral sinuses, may produce cerebral symptoms of back pressure.

The importance of the lateral sinus in otitic sinus thrombosis may be overstressed, since it may cloud the diagnosis when thrombosis of the sagittal veins, the petrosal veins, the jugular bulb or the cavernous sinus may exist.

The importance of the petrosquamous sinus in tympano-antral infections has been overlooked according to some authorities. Perhaps this is due to the fact that this sinus does not often persist in the adult. In infancy and childhood, however, it should always be considered, since it brings about the anastomoses of the veins of the middle ear and the meninges. Blood and inflammatory products will be carried directly into it from tympano-antral suppuration. This frequently results in an endocranial lesion without macroscopic changes in the mastoid bone.

The surgical importance of the mastoid emissary vein should always be borne in mind. When present, this vein may be primarily infected from disease of the temporal bone or secondarily involved from thrombophlebitis of the lateral sinus. Law [80] stressed the importance of this vein in its relation to the size of the lateral sinus and stated that the size of this sinus cannot be determined unless the entrance of the emissary shows.

A thrombus is essentially a protective measure and is not, as the minority believe, infected from the beginning. It is nature's method of blocking the blood channel in an effort to prevent further spreading of an infection. It is the secondary infection of the clot and not the thrombus that constitutes the danger.

Atypical symptoms associated with endocranial disturbances may be due to anomalies of the sinus structure, mild infections, cachectic or debilitating conditions, the low virulence of the micro-organism, involvement of the jugular bulb or the petrosal veins or a sudden occlusion of the larger sinus or jugular vein, causing obstruction of the superficial cerebral veins, thus producing venostasis of the cortex.

From a series of corrosion preparations, Batson has demonstrated many intercommunications of the venous system, with extensive endocranial ramifications which do not lend themselves to any surgical treatment yet devised. Careful study of these preparations helps one to comprehend more clearly and to visualize the anatomic arrangement and distribution of the intracranial venous system and what may happen inside the cranium and the course an inflammatory lesion may be likely to take in its passage through the dura from disease of the temporal bone.

CONCLUSIONS

From a comprehensive review of the literature based on the physiologic, anatomic and pathologic conditions involved in the causation, production and distribution of suspected thrombosis of otitic origin and from the study of the records of past clinical experiences, it appears that no unified surgical procedure can be adapted for this condition other than the eradication and drainage of the primarily infected area. The use of surgical intervention on the jugular vein is still controversial.

While great advances have been made in the diagnosis and treatment of sinus thrombosis, much still remains to be desired in treatment in accomplishing a complete eradication of the infective foci; yet through every rift of discovery along the pathway of progress in this complex field one should keep in mind that eternal dissatisfaction is the price of progress.

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80. Law, Fredrick M.: Personal communication to the author.

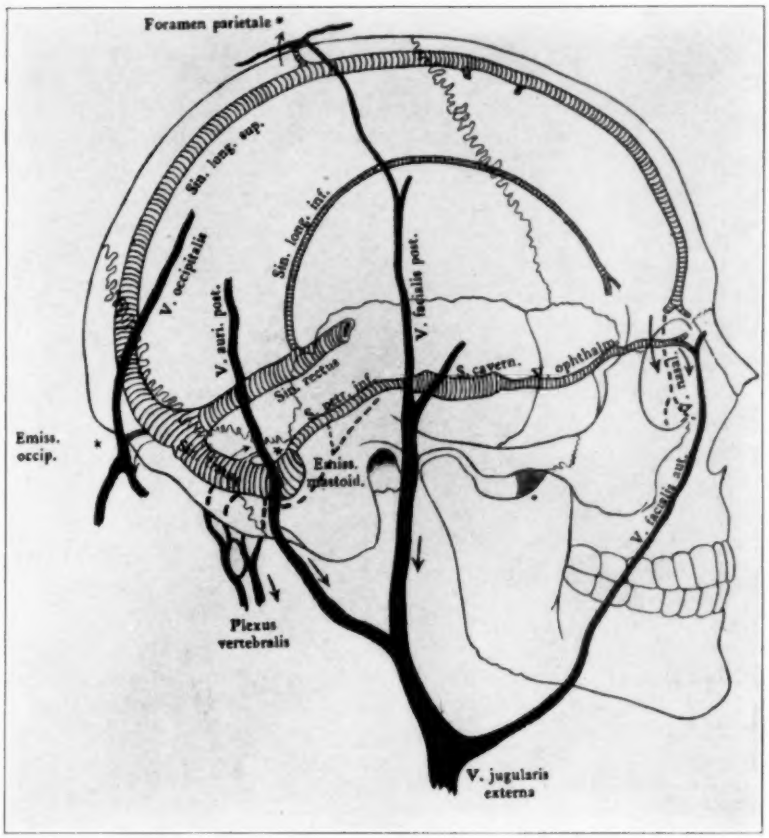

Fig. 1.—The sinuses and veins of the cranium. (Reprinted from Ballance and Green[7]; original by Hermann and Merkel.) Lateral view of the face and skull, showing the communication of the surface veins with the endocranial sinuses through the medium of the principal emissary veins.

The veins of the scalp join the veins of the face and neck, especially the external jugular vein. They all communicate with the sinuses of the dura mater and indirectly with the veins of the pia-arachnoid by means of innumerable small emissary veins which pass through the skull. In addition, there are a number of larger ones:

1. Between the frontal vein and the longitudinal sinus.
2. Between the nasal vein and the cavernous sinus, through the ophthalmic veins.
3. Between the posterior auricular veins and the lateral sinus (mastoid).
4. Between the occipital veins and the lateral sinus.
5. Passing through the parietal foramen, the occipital foramen, the foramen magnum and the numerous openings in the base of the skull.

Not infrequently infection will spread from the skin and subcutaneous tissue of the face or scalp and cause thrombophlebitis of the subcutaneous veins, which may extend through the emissary veins into the interior of the skull, causing septic thrombosis of an endocranial sinus or septic meningitis.

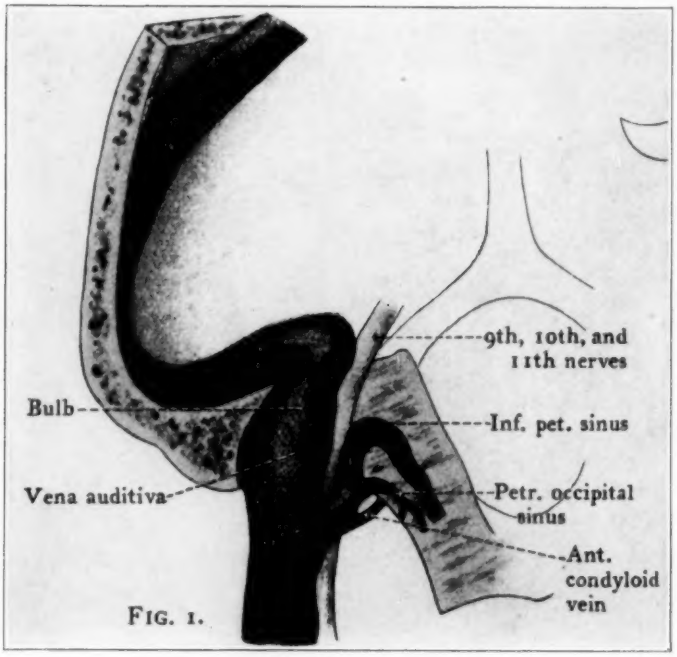

FIG. 1.

Fig. 2.—Front view of the right jugular bulb. (From Charpy, in Poirier, P., and Charpy, A.: Traité d'anatomie humaine, ed. 3, Paris, Masson & Cie, 1912. Reprinted from Ballance and Green.[7]) The venous connections of the jugular bulb are important and have been described in considerable detail by Macewen and by several German authors, notably Hansberg and Zuckerkandl.

They are:

1. The sinus petrosus inferior.
2. The vena aqueducti cochleae.
3. An anastomotic communication with the occipital sinus by a vein which traverses the base of the occipital bone.
4. The posterior condyloid vein, usually a large vein, connects the bulb with the spinal plexus outside the dura.
5. The anterior condyloid vein, which is constant and connects the bulb with veins within and without the skull.

Thus the jugular bulb is in venous connection with the cavernous sinus, the internal ear and the venous plexuses in the cerebellar fossa, in the spinal canal and beneath the basisphenoid. The jugular bulb varies considerably in size and in its exact relation to the tympanum. It sometimes attains the size of a large cherry and is sometimes scarcely noticeable. In certain cases it is necessary to expose and incise it fully in order to remove a dangerous focus of suppuration or as part of the operation for septic thrombosis of the cavernous sinus.

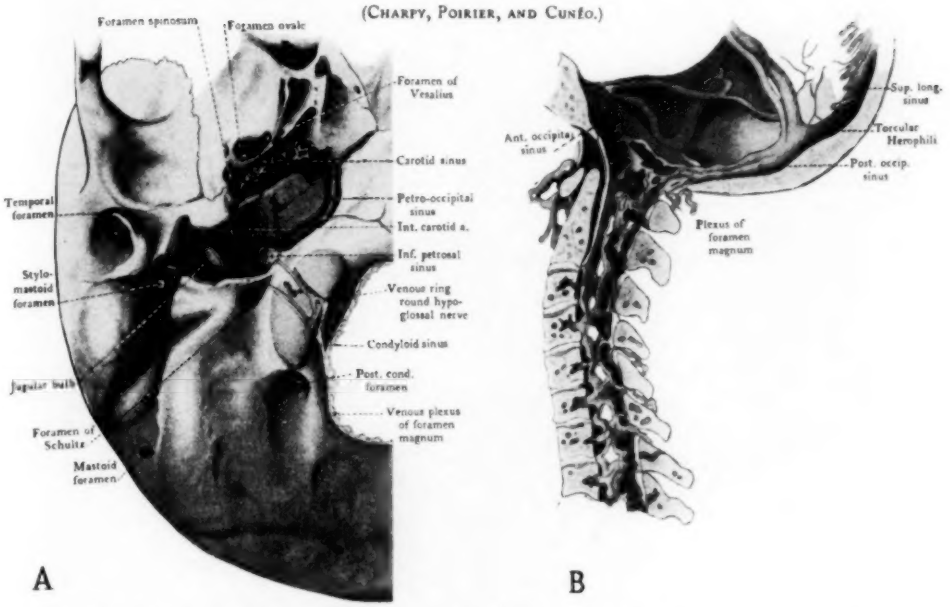

Fig. 3.—Some veins at the base of the skull. *A*, the posterior portion of the right half of the base of the skull, showing the foramina and the venous channels. (Reprinted from Ballance and Green.[7] Original by Charpy, in Poirier and Charpy: Traité d'anatomie humaine.) *B*, sagittal section of the upper part of the spinal column and of the posterior fossa of the skull, showing the anastomoses between the spinal venous plexus and the cranial sinuses. (Reprinted from Ballance and Green.[7] Drawn by Charpy, after Breschet.)

Under normal conditions almost the whole of the blood from within the cranium is returned by the two internal jugular veins; there are, however, numerous anastomoses between the intracranial and the extracranial venous circulation which allow alternative routes when one or the other of the jugular veins is not available. These venous communications are sometimes also the channels through which infection from without reaches the interior of the skull and vice versa.

There are three principal and many accessory communications. The three principal ones are: (1) The plexus of the foramen magnum, the uppermost of the spinal plexuses, anastomoses with the transverse occipital sinus in front and with the posterior occipital sinuses behind. (2) The ophthalmic vein and its affluent veins, through which a considerable volume of blood may enter the facial and temporal veins. (3) The middle meningeal veins, through which there is communication between the superior longitudinal sinus and the pterygoid plexus.

The accessory communications are innumerable, for the origins of the external jugular vein and of the facial portion of the internal jugular vein communicate with those of the intracranial veins practically everywhere, over the vault and over the base.

The true emissary veins are all situated on the vault of the skull. They are: (1) The parietal emissary vein, the mastoid emissary vein, the occipital emissary vein and the emissary of the foramen caecum.

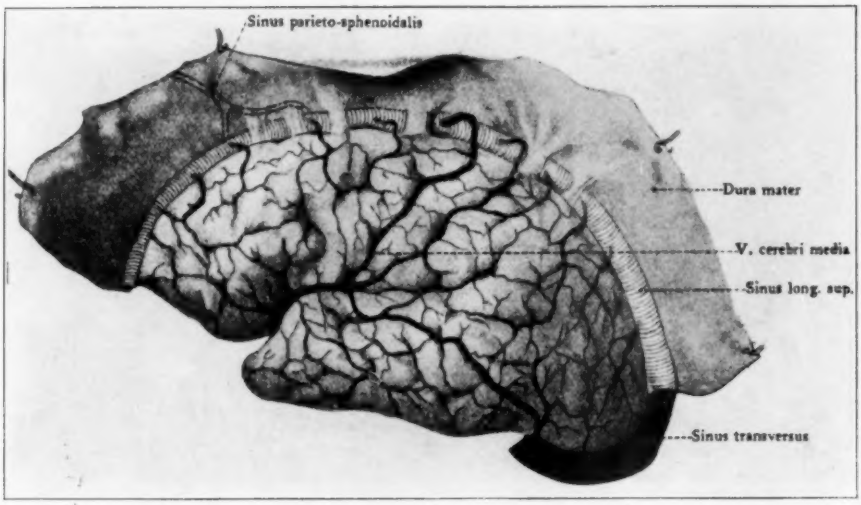

Fig. 4.—The veins of the convex surface of the cerebral hemisphere (reprinted from Ballance and Green.[7] Original by Hermann). The venous blood from the cerebral hemispheres is collected by a number of veins of varying size, which empty into the superior longitudinal sinus at short intervals throughout its course. The openings of these veins into the sinus are at right angles or face obliquely forward, so that the current of blood from the veins is against that in the sinus. One will notice also their connections with the lateral sinus.

The cerebral and cerebellar veins which empty into these more rigid dural sinuses possess no valves and have extremely thin walls, owing to a lack of muscular tissue. They pierce the pia-arachnoid membrane and the meningeal layer of the dura before they reach the sinuses, thus exposing the subarachnoid and subdural spaces to infection in case the sinus becomes involved from thrombophlebitis. It is these anatomic intercommunicating channels of the lateral sinus that may give rise to some of the anomalous pathologic manifestations observed after otitis media or mastoiditis.

Thrombosis, particularly marantic thrombosis in childhood, sometimes extends from the sinus into these veins, and when certain veins are involved focal symptoms may arise, leading to the suspicion of abscess or tumor.

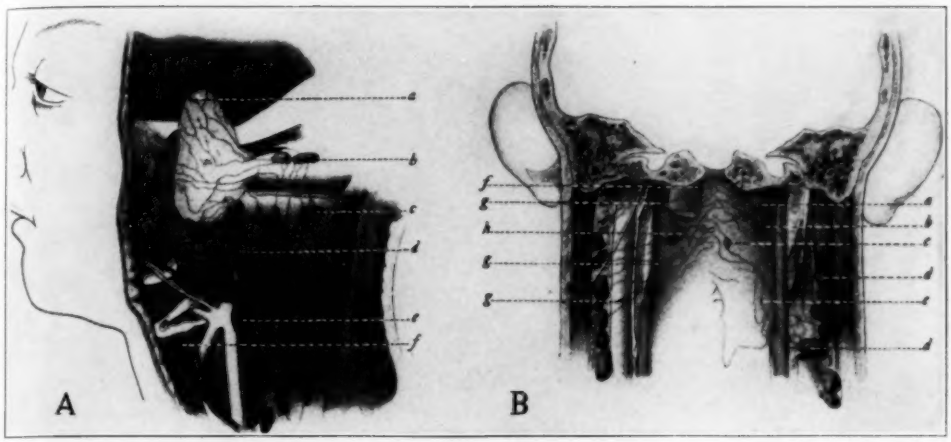

Fig. 5.—*A*, the deep cervical glands of the pharynx.

a, lymphatics of pinna

b, mastoid glands

c, sternomastoid glands (external group)

d, gland of the external jugular chain

e, sternomastoid gland belonging to the inner group of the internal jugular chain

f, aberrant subhyoid gland placed in the track of the efferent vessels of the submental glands

a and *b*, retropharyngeal glands

c, intermediate gland placed in the track of the efferent vessels from the retropharyngeal glands which pass in front of the internal carotid artery

d, gland belonging to deep cervical chain

e, efferent vessels from the retropharyngeal glands passing in front of the internal carotid artery

f, afferent vessels of the retropharyngeal glands passing in front of the rectus anticus

g, lymphatics from the pharynx passing directly into the deep cervical glands

h, afferent vessels of the retropharyngeal glands

(Reprinted from Ballance and Green.[7] Original in Poirier, P., and Cuneo, B.: Abrégé d'anatomie, Paris, Masson & Cie., 1909.) The upper group of deep cervical glands extends from the base of the skull to the bifurcation of the carotid artery and is in contact with the internal jugular vein. In septic thrombosis of the sigmoid sinus these glands become enlarged and tender, and before the incision is made in the neck, the tender gland cord may be mistaken for a thrombosed distended jugular vein. When the thrombosis does not extend beyond the bulb, the vein is usually found collapsed and like a fibrous cord.

The retropharyngeal glands are specially liable to infection in young children. The swelling causes dyspnea and dysphagia. These conditions must not be confused with spinal abscess or conditions of the larynx requiring tracheotomy.

B, the lymphatics of the pinna (internal surface). (Reprinted from Ballance and Green.[7] Original in Poirier, P., and Cuneo, B.: Abrégé d'anatomie, Paris, Masson & Cie., 1909.) The collecting trunks from the pinna may be divided into three groups: posterior, anterior and inferior.

Abscess of the mastoid gland must be differentiated from subperiosteal abscess and from a purulent collection escaping onto the mastoid region from the meatus. In inflammatory infection of the meatus and the deeper parts of the temporal bone the glandular involvement is determined by the anatomic disposition of the lymphatics, as illustrated in this figure.

Fig. 6.—The internal jugular vein. (From Ballance and Green.[7] Original by Spalteholz.) The deep veins of the neck viewed from the right. The muscles of the neck and face have been in large part removed; the bones of the face have been partly removed.

In infective thrombosis of the sinus sigmoideus the coagulum has been observed to spread as far as the right auricle. I have on several occasions seen the jugular vein thrombosed as far as the valve at the upper limit of the bulbus venae jugularis inferioris and once as far as the vena subclavia. In one case death was caused by spasm of the glottis, the thrombosis having extended from the jugular vein into the vena laryngea superior.

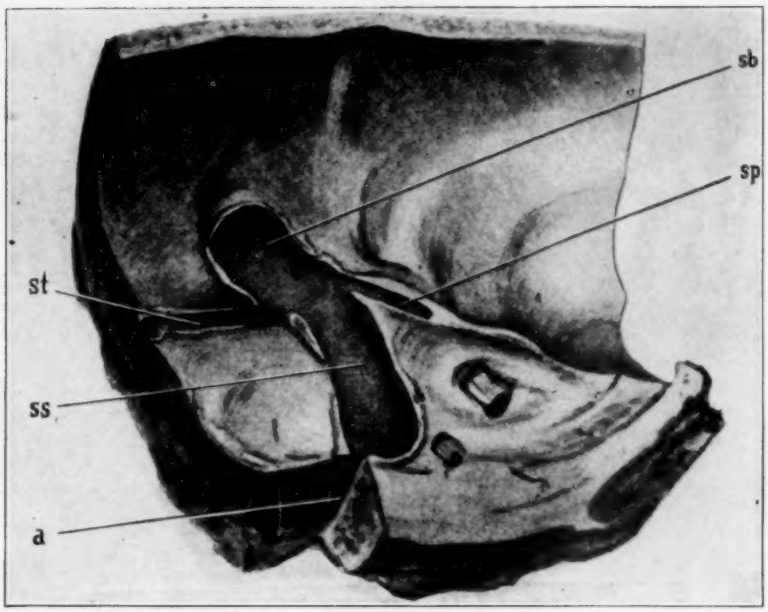

Fig. 7.—Anomaly of the left lateral sinus. This picture is self-explanatory. It shows a blind sac (*sb*) at the knee of the sigmoid portion of the lateral sinus. The following abbreviations are used: *st*, sinus transversus; *ss*, sinus sigmoideus; *sp*, sinus petrosus superior, and *a*, a narrow passage to the vena jugularis interna.

This picture was sent to me by Prof. Erich Ruttin in a personal communication. It originally appeared in his article.[20]

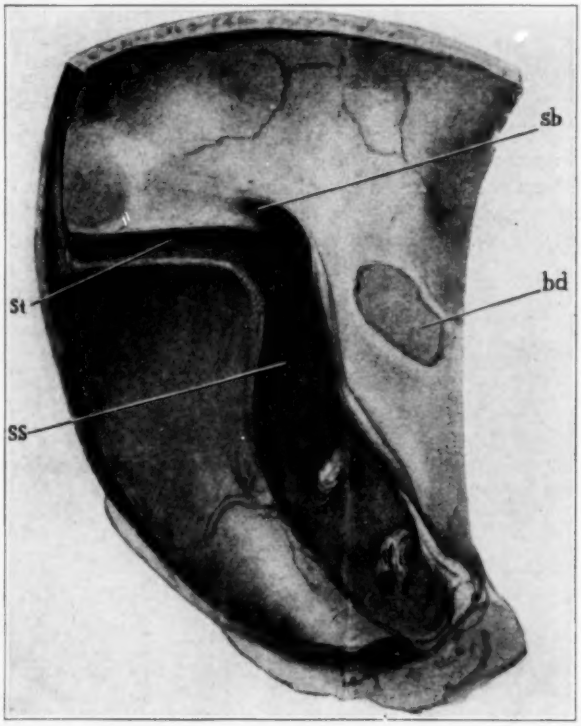

Fig. 8.—Anomalous condition of the left lateral sinus. This condition is described in the text of the thesis. The picture is self-explanatory. The abbreviations used are as follows: *st*, sinus transversus; *ss*, sinus sigmoideus; *sb*, blind sac, and *bd*, the operative closing of the defect in the tegmen.

This picture was sent to me by Prof. Erich Ruttin in a personal communication. It originally appeared in his article.[20]

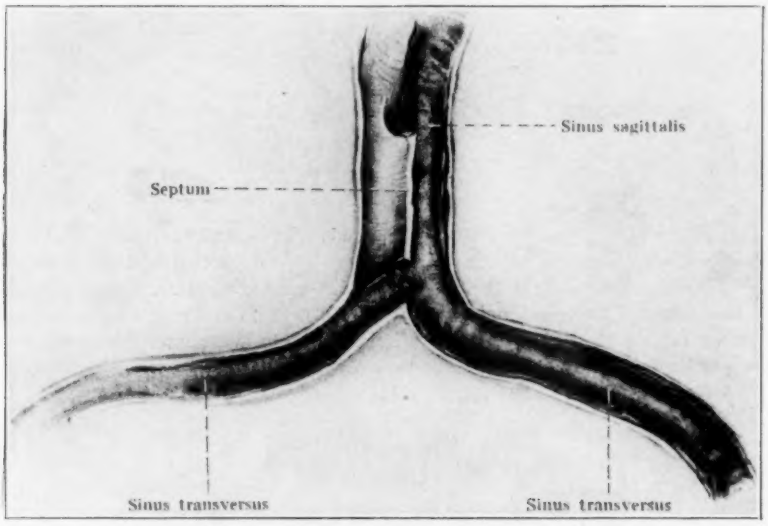

Fig. 9.—This illustration represents an intermittent, or propagated, thrombosis of the right sigmoid and transverse sinuses which extended directly into the superior sagittal sinus. With a sound, Ruttin did not come into the torcular Herophili at the operation, but over the region of the torcular directly into the superior sagittal sinus. The transverse sinus of the left side enters the sinus rectus.

Through their researches Henrici and Kikuchi found only 3 per cent of the specimens in which the superior sagittal sinus arose from the right transverse sinus while the left transverse sinus arose from the sinus rectus. Ruttin stated that this anatomic condition is much more frequent than Henrici and Kikuchi assumed.

The clinical history of this case and the original of the figure were published by Ruttin (Zur Klinik der intermittierend fortschreitenden Thrombose des Sinus transversus und sagittalis, Acta oto-laryng. **19**:66, 1933).

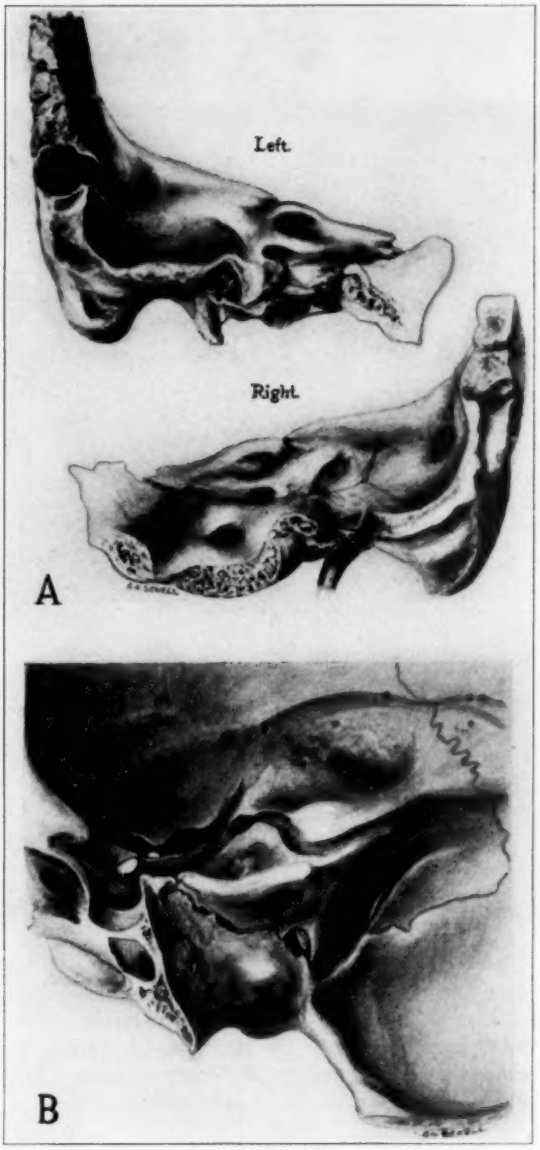

Fig. 10.—Anomalous condition of the lower part of the sigmoid sulcus. (Reprinted from Cheatle.[9]) These illustrations show the atavistic abnormality in which the mastoid emissary vein takes the lower part of the sigmoid sulcus.

The left side of figure *A* shows a sigmoid sulcus terminating in the large mastoid foramen. A similar condition is met with on the right side.

B represents a similar condition which was noted on the left side in which the sigmoid sulcus terminated in the large mastoid foramen.

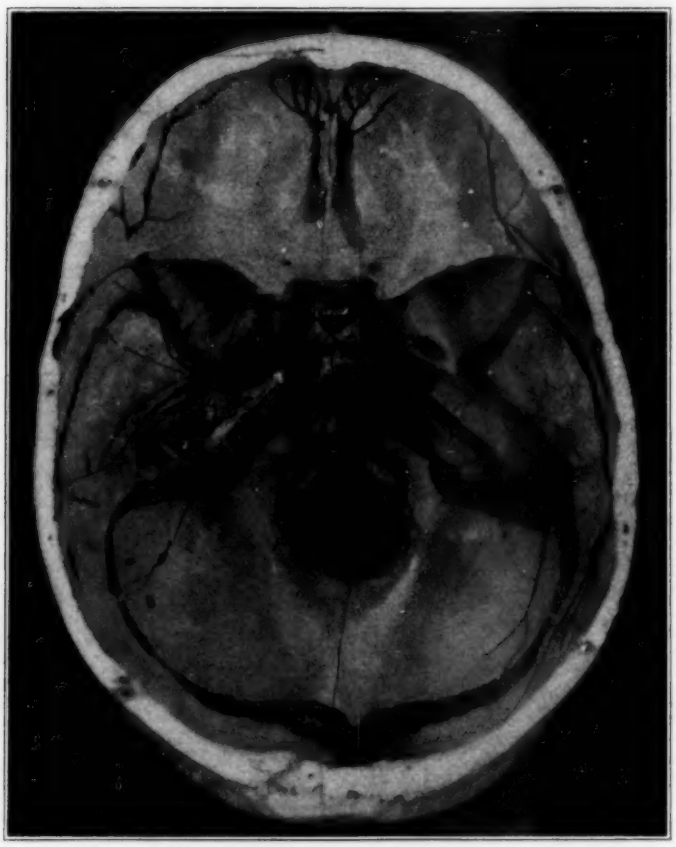

Fig. 11.—A view of the inner base of a skull owned by me. The internal view of the base of the skull shows a great difference between the size of the two lateral sinuses, the right being much the larger of the two, especially the sigmoid sulcus. The sulcus of the sigmoid portion of the right lateral sinus is deep and extends up toward and under the overhanging lip of the petrous apex. The greatest width of the sigmoid sulcus on the right side is 1.5 cm., while on the left side, the greatest width is 7 mm.

The sinuses forming a network separating the petrous apex from the cerebral cavity are well shown on the markings of this skull.

The significance of the pathologic conditions of the temporal bone in its relation to this network of sinuses is well appreciated by the otologist.

Fig. 12.—Lateral internal view of the right side of the skull. From a skull owned by me. The sulcus for the right lateral sinus is deep, especially for the sigmoid portion. The knee of the sigmoid extends upward and under the overhanging lip of the posterior part of the petrous apex. This also shows the internal auditory meatus and the position of the inferior and superior petrosal veins and the cavernous sinuses in relation to the petrous apex.

This view also shows the ramification of the middle meningeal artery and its branches.

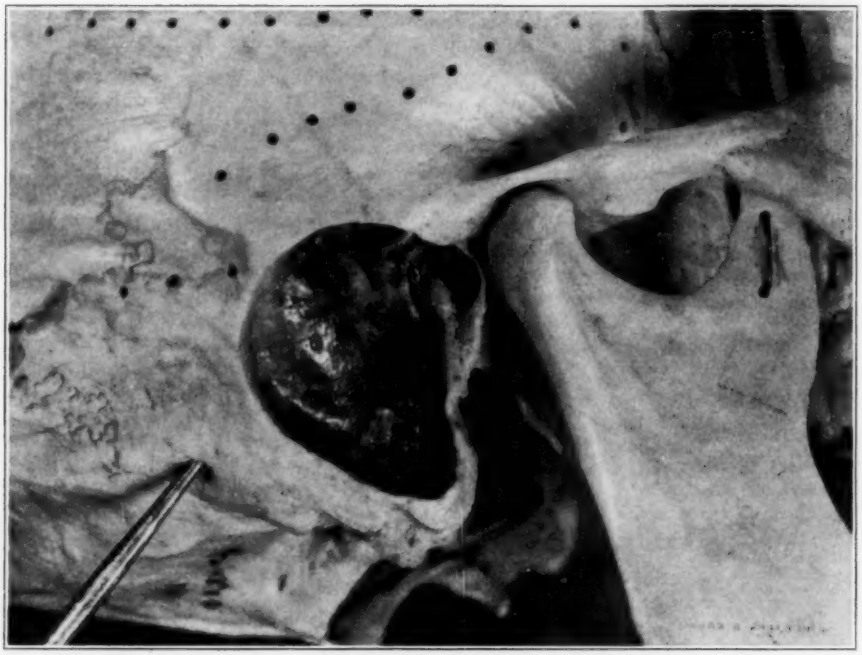

Fig. 13.—The external surface of the right side of the skull. From a skull owned by me. This picture shows a complete evisceration of the mastoid and opening of the semicircular canals. The sigmoid portion of the lateral sinus is superficial; the cortex is about 3 mm. thick. The lateral sinus bulges into the mastoid cavity and is about 2 cm. wide. The facial canal has been exposed and is represented by a probe. A large mastoid foramen, 6 mm. wide, is present.

In the base of the skull is a large jugular fossa, which is about 2 cm. deep and about 11 mm. wide on the right side. The fossa is practically absent on the left side. The jugular foramen on the right side is more than twice the size of the one on the left.

OSTEOMYELITIS OF THE MANDIBLE

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DENVER

The mandible differs from other bones of the body in that it is exposed on all sides and consequently is open to injuries and infections from any or all directions. Infection may travel through a tooth pulp or socket directly to the interior of the bone. On the other hand, hematogenous infection of the mandible or infection along a nerve trunk is probably no different from that in other bones. Osteomyelitis of the mandible probably resembles osteomyelitis following sinus disease in a bone of the skull or in some other bone of the face. Much has been written by Mosher, Fuerstenberg and others on etiology, pathology and treatment of osteomyelitis following sinus disease, and these writings apply equally well to the subject of this paper.

The etiology is so well understood that, aside from the fact that there are undoubtedly certain patients who are predisposed toward osteomyelitic infection, osteomyelitis of the mandible should be one of the avoidable diseases. The causative factor is odontogenous or traumatic, such as a fall, a blow, a gunshot wound or operative manipulation. It is doubtful which ranks first as a forerunner of the condition, dental caries, or fracture, dental extraction and other traumatic procedures. Among the less frequent causes are the exanthematous fevers, osteomyelitis elsewhere in the body, other surgical procedures, radium treatment of the bone, spasm of the jaw muscles, tuberculosis, and typhoid. Among the possible traumatic causes is block anesthesia, during which injury to the vessels may occur. In many of my cases the condition followed operation for removal of teeth by this method. Suture of wounds which may be infected or failure to drain wounds which may close over a blood clot that already is or may become infected may also be causes. In one of my cases, involvement followed cleaning of the teeth by a dentist.

In osteomyelitis, teeth may be involved even though the roentgenogram shows no evidence, because the lingual and buccal aspects of the tooth are not visible on the film. Obviously, it is impossible to portray those aspects with any of our present methods. Mead [1] wrote that the

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Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 9, 1937.

1. Mead, Sterling V.: Diseases of the Mouth, St. Louis, C. V. Mosby Company, 1932.

"majority of cases of osteomyelitis of the mandible and maxilla are caused by an extension of the infection from a carious tooth, from a gangrenous pulp of an apparently sound tooth, from an alveolus after an extraction, or from a wound reaching the bone or periosteum, as is most commonly seen in fractures of the mandible. Naturally, those caused by caries are by far the most common."

As to frequency of occurrence: In 1932, Wilensky [2] reported that statistics showed that of the bones affected by osteomyelitis the jaws were the eighth in frequency and that the lower jaw was much more often affected than the upper one. A higher incidence in adults is probably due to the greater amount of surgical intervention done today on third molar teeth, impacted or otherwise, in which they are removed. It is also traceable to the surgical technic used by many dentists in which they sew flaps of tissue after the extraction and include infectious material in the cavity. In the analysis of 234 cases reported by Brown and Tung [3] in 1936, the site of the disease was found to be the maxilla in 48 cases and the mandible in 186. Males are more frequently affected than females; the usual proportion stated is about 3:1. Conner [4] stated the belief that the increased incidence in men was due to their inattention to instruction and their failure to cooperate following dental work.

The disease occurs in persons of all ages, but the analysis of Brown and Tung [3] of their cases indicates that the greatest incidence is between the ages of 21 and 40 years. The condition in children has been differentiated from that in adults. In the former it is a secondary disease and occurs sometimes as a result of an acute exanthematous fever. Wilensky [2] wrote that the only cause for differentiation is that there is more spongiosa in the bone of the young, and that as long as there is growth there is greater blood and lymph supply. Consequently the younger the subject the more frequent the occurrence of hematogenous or odontogenous osteomyelitis. However, there is not as much decay of the teeth in children as there is in adults. Therefore the frequency mentioned is offset by a relatively lower number of causes in children. Wilensky [2] also stated that in young children the upper jaw is more often affected than the lower. I question that statement, as my experience has been just the reverse. Carr,[5] discussing chronic periapical

2. Wilensky, A. C.: Osteomyelitis of the Jaws, Arch. Surg. **25**:183-237 (July) 1932.

3. Brown, James, and Tung, P. C.: Osteomyelitis of the Jaws, Internat. J. Orthodontia **22**:69-80 (Jan.) 1936.

4. Conner, Thomas: Osteomyelitis of the Mandible, J. Am. Dent. A. **22**: 1190-1193 (July) 1935.

5. Carr, Malcolm W.: Diseases, Malformations and Injuries of the Mouth and Associated Parts Occurring in Children, Internat. Clin. **4**:233-243 (Dec.) 1933.

disease (usually streptococcic) extending beyond the apex of the root of the tooth in children, recognized four stages, any or all of which might constitute active primary foci capable of producing various secondary lesions, among which was osteomyelitis. These four stages are: (1) chronic proliferative pericementitis; (2) chronic rarefying osteitis with granuloma; (3) chronic rarefying osteitis with suppuration, and (4) chronic rarefying osteitis with cyst formation.

There does not seem to be any general agreement as to a particular organism predominating in the pus. Brown and Tung [3] analyzed one group of their cases and found that the following becteria were isolated: Streptococcus haemolyticus in 7 instances; Staphylococcus aureus haemolyticus in 5; Staphylococcus albus haemolyticus in 2; Staph. aureus in 4; no growth in 5; a diphtheroid bacillus in 1; the tubercle bacillus in 2; Actinomyces in 3, and a mixture of organisms in 12. There was a positive Wassermann reaction in 15. In addition, we have frequently found Vincent's organism in smears. This is not considered pathogenic except when found in large numbers.

The disease is important because of the discomfort it affords the patient and because of the possible complications. According to Wilensky,[2] the latter may be due to lymphatic drainage, a general infection (bacteremia, sepsis, septicemia), or a local condition, such as abscess, ankylosis, phlebitis (rare and usually on the side opposite the previous operative procedure), meningitis (also rare), cerebral abscess, erysipelas or tetanus. The last named condition is probably confused with trismus. The floor of the mouth and the salivary glands are often involved in cases of osteomyelitis of the mandible, which is probably a very common condition, although in 1912 Blair [6] stated that he had oberved but a single case of acute metastatic osteomyelitis of the jaw bone. There is also the problem of lifelong defects. This is especially important in children. The case reported by Ivy and Curtis [7] in January 1936 illustrates the importance of this point. The patient was a woman, 27 years old, who had had osteomyelitis of the mandible at 7 years of age. Her chin was left badly distorted. When the authors saw her she had already lived twenty years with the deformity and to correct it necessitated a bone graft and an artificial denture. The result was not perfect, although the cosmetic effect was improved.

It is the dentist or the otolaryngologist who is most often sought first by the patient with a slight oral lesion or evidence of distress, and therefore it is essential that both of these specialists be equipped to

6. Blair, V. P.: Surgery and Diseases of the Mouth and Jaws, St. Louis, C. V. Mosby Company, 1912.

7. Ivy, Robert H., and Curtis, Lawrence: Loss of Substance of the Mandible Following Osteomyelitis, Ann. Surg. **103**:149-152 (Jan.) 1936.

handle an examination which will elicit the proper diagnosis and permit inauguration of the proper treatment.

The diagnosis of osteomyelitis may be comparatively easy, as it is characterized by swelling, tenderness, loosening of the teeth, at times welling up of pus, and pain on percussion. The patient may complain of deep boring pain such as occurs in the long bones, but this is always before drainage of pus takes place. One important point which is not stressed in studies or discussions of osteomyelitis of the skull, the sinuses or the jaws is that the roentgen indications are of little value before the eighth to the tenth day, because the changes in the bone are not sufficient to interfere with the passage of the rays. Symptoms of Ludwig's angina are present in most cases of osteomyelitis of the mandible. Wolfe [8] was of the opinion that osteomyelitis of the body of the mandible differs from that of the ramus in its clinical appearance and prognosis. He emphasized hypoesthesia of the lip as an early important diagnostic sign of involvement of the body. This is due to pressure in the canal caused by swelling, but I have seen some cases in which it was due to injury of the nerve by a penetrating spicule of bone during or after extraction.

The treatment is generally conceded to be establishment and maintenance of drainage, with an interval to permit formation of sequestrums, then removal of all remaining sequestrums, and attention to healing and the end result. In 1934 Miltner and Wolfe [9] proposed what they called a more radical procedure, which they had used successfully in 8 cases. It embodies several steps: (1) removal of the necrotic outer plate of the mandible as soon as possible during the early chronic stage (that is, twenty-one days after the onset of disease) through a wide external approach (even before sequestrum formation); (2) removal of teeth over the area of involvement; (3) exteriorization of the tooth sockets and partial resection of the alveolar process with complete closure of the gum margins to prevent further drainage of the purulent material into the oral cavity, and (4) immobilization of the jaw. In 1936, Chalmers Lyons [10] reported his use of a similar radical therapy, which in his opinion was of value because it shortened the period of convalescence and the duration of the disease. He stated the belief that it prevented pathologic fracture and checked continued extension of the infection and the sequestration. He employed appliances, fracture bands, rubber bands, stainless steel pins and other apparatus for main-

8. Wolfe, J. J.: Osteomyelitis of the Mandible: Observations on Etiology, Pathogenesis and Diagnosis, Chinese M. J. **49**:422-428 (May) 1935.

9. Miltner, Leo J., and Wolfe, J. J.: Treatment of Suppurative Osteomyelitis of the Mandible, Surg., Gynec. & Obst. **59**:226-235 (Aug.) 1934.

10. Lyons, Chalmers: A Radical Treatment for Chronic Osteomyelitis, J. Am. Dent. A. **23**:1092-1095 (June) 1936.

taining physiologic rest. On the other hand, Brown and Tung [3] found the conservative procedure to be wise, especially as far as attacks on the bone itself were concerned. Their opinions are especially valuable because their report was carefully compiled and it represented careful procedures carefully controlled and supplemented with proper bacteriologic studies and the use of laboratory aids when indicated.

The conclusions drawn in this report are based on a study of 86 cases observed in the period from 1927 to 1937. I prefer a method of treatment which embraces the following steps: (1) establishment and maintenance of drainage, preferably externally, at the lowest point for relief of congestion; (2) removal of teeth apparently abscessed or loosened; (3) allowance of an interval for sequestrum formation and (4) removal of sequestrums and attention to healing. I guard against involucrum formation and when indicated stimulate cell growth by the assistance of radiation therapy.

Because dental caries is such an important factor in the production of osteomyelitis, the physician must add the knowledge of its importance to his mental awareness in examining the oral cavity. Recent dental periodicals are replete with material designed to teach dentists how to recognize the early evidences of oral infections which are potentially dangerous to the patient. We physicians must be equally acute in such inspection. There is nothing new in the statement that teeth and tonsils may harbor foci of infection which are potential sources of cardiac disease, arthritis, septicemia and blood dyscrasia. The dentist is no longer satisfied with mere inspection of the teeth for caries and gingivitis, or even with the added information afforded by a roentgen examination of the entire mouth. He now seeks detailed information as to the existence of infection around the pyorrheal tooth. Because this tooth may have more than one root with more than one abscess around each root, and because it has little bony support, it is important as an etiologic factor in osteomyelitis. The otolaryngologists, especially if they are interested in oral surgery, must be as keenly observant as are the dentists.

ABSTRACT OF DISCUSSION

DR. V. H. KAZANJIAN, Boston: The essayist has adequately described osteomyelitis of the mandible in all its essential points. It is a disease that in its early stage may be alarmingly serious and may even lead to fatality. The convalescing stage is slow and often lasts months and not infrequently leads to permanent deformity.

As pointed out in the paper, the vast majority of cases are caused (1) by dental infections and (2) by trauma.

Osteomyelitis of the mandible from other causes mentioned in the paper is extremely rare. I have observed only one case in which the disease might have had a hematogenous origin. This case was that of a baby 6 months of age who suffered a definite infection of the mandible in the left premolar region. Radiation

necrosis is seen more often. In such cases sequestration occurs slowly, assuming an extremely chronic course.

A study of 100 cases at the Massachusetts General Hospital in 1926, made by Miner, showed that 75 per cent of the patients had a history of dental disturbances previous to the onset of the disease. Toothache followed by filling of teeth or removal of teeth was the common finding. Brown and Tung, in their study of 234 cases cited in Dr. Carmody's article, gave approximately the same percentage. My clinical observations lead me to think that the estimates given are conservative, even though they seem high.

A further analysis of this group of cases brings out two outstanding points: 1. Osteomyelitis of the mandible occurs most frequently in patients who have neglected the proper care of their teeth, patients with caries or pulp involvement, with areas of rarefying osteitis on pulpless teeth or with pyorrhea pockets. These are the foci from which organisms invade the bony structures. 2. There is a high incidence of osteomyelitis following dental and surgical operations on and about the teeth. This leads to the supposition that operative trauma might have precipitated the spread of infection into the bony structure.

For the minimization of the occurrence of osteomyelitis of the mandible, responsibility seems to be divided between the patient and the operating dentist or surgeon. The physician can do little toward the care of the patient's mouth except to give good advice and to try to educate him against the dangers of neglected hygiene of the mouth. If certain surgical and dental procedures often lead to precipitation of bone infection, they should be investigated and the technic modified.

There is no doubt that in all intraoral surgical operations careful maintenance of asepsis and delicate handling of tissues will minimize operative trauma. In the treatment of acute infections of the mouth it is important to use good judgment as to the time and the extent of surgical intervention. It is a debatable question whether or not in the presence of acute infection of a tooth the offending tooth should be extracted. It was an accepted rule with dentists of past generations never to remove a tooth until the infection was localized. This rule was abandoned by the younger generation, only to be revived again in a modified form recently.

Acute infections originating in the dental pulp or pericemental membranes cause hyperemia and congestion in their earliest stage and gradually permeate the deeper tissues. Removal of the offending tooth will relieve the congestion if the lesion is still superficial and the alveolar socket offers sufficient drainage. It is in this stage that most threatening abscesses are aborted. If, however, the inflammation has invaded the deeper structures, it is obvious that the removal of the tooth will not give adequate drainage; on the contrary, it may aggravate the inflammation by causing more trauma. The proper treatment, therefore, is to disregard the tooth temporarily, direct all the attention toward localizing the inflammation and operate as soon as the presence of free pus is suspected.

Fracture of the mandible in a certain number of cases results in localized osteomyelitis and eventual nonunion of the fragments. The main causes of this complication are (1) the type of injury inviting the micro-organisms of the mouth to invade the deeper structures; (2) the presence of isolated bone fragments and other foreign bodies introduced during the accident and, most frequently, (3) the presence of nonvital teeth in the line of fracture. Here again good judgment as to the time and the extent of surgical intervention will prevent many complications. In a case of fracture, early surgical intervention, such as

removal of a foreign body or of broken-down teeth in the line of fracture and early immobilization, will go a long way toward producing an uneventful consolidation of the fractured bones.

I should like to emphasize the importance of preventing or minimizing the occurrence of osteomyelitis in children. The disease is far more serious in children than in adults, owing to the fact that it leads to permanent deformities much more frequently.

DR. ROBERT H. IVY, Philadelphia: There is one avenue of infection occasionally leading to osteomyelitis of the mandible that Dr. Carmody did not mention, namely, a furuncle of the chin. Osteomyelitis of the anterior part of the mandible may cause a localized suppuration of the soft tissues simulating a furuncle and thus give rise to confusion, but I think that I have seen 2 true skin furuncles in the region of the chin from which infection has extended into the bone. G. Hesse published a paper on this subject (*Deutsche Monatschr. f. Zahnheilk.* **12**:529, 1933).

I am in agreement with Dr. Carmody that one should be conservative in the treatment of osteomyelitis, providing adequate drainage and waiting until sequestrums form before doing a great deal to the bone. I think that it is possible to save a great many teeth which appear to be hopelessly loosened by the disease but which if allowed to remain will sometimes tighten up and become useful.

I wish particularly to emphasize the secondary deformities resulting from osteomyelitis of the lower jaw. Much can often be done while the disease process is going on, by means of splints on the teeth, to hold in proper position the healthy part of the jaw and thus to avoid shortening of the bone with malocclusion of the teeth and external disfigurement. After deformity has occurred, it may be possible to restore the jaw to its natural length and thus reestablish occlusal relationships by bone grafting.

DR. THOMAS E. CARMODY, Denver: I wish to thank Dr. Kazanjian and Dr. Ivy for their discussion. One of my slides, which I was unable to exhibit, showed a pathologic fracture of the type of which Dr. Kazanjian spoke. His method of taking care of the dead teeth is to be complimented. Also, I think that what he said about waiting to remove teeth in cases in which one thinks that they are loosened and feels that it is necessary to remove them is something that one should always keep in mind, because many of those teeth can be saved. Also, from the standpoint of roentgenology one must keep in mind that roentgen examination does not show the full pathologic significance of the disease unless made after the eighth or the tenth day.

Instead of depending on clinical judgment, some years ago I waited for the roentgenogram to show me where the trouble was and the extent of it. Instead of taking out two teeth I had to take out eight.

I want to thank Dr. Ivy for mentioning the furuncle of the chin. I have never observed a case in which I thought the condition was due to that cause. Mentioned in the paper was a case, also observed by Dr. New, of osteomyelitis of the inner plate of the mandible caused by a little abrasion made during simple cleaning of the teeth.

Dr. Ivy is ingenious in his method of holding the space. I have a patient under treatment at the present time in whom we are holding the space by means of orthodontic appliances, as he suggests. A plastic surgical procedure has to be done in many of the cases.

I compliment Dr. Ivy on the results that he has shown in the cases in which he has operated.

CARTILAGE TRANSPLANTED BENEATH THE SKIN OF THE CHEST IN MAN

EXPERIMENTAL STUDIES WITH SECTIONS OF CARTILAGE PRESERVED IN ALCOHOL AND BURIED FROM SEVEN DAYS TO FOURTEEN MONTHS

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This experimental work was performed to determine the fate of human costal and septal cartilage preserved in alcohol and transplanted beneath the skin of other human beings.

The use of cartilage preserved in alcohol to fill depressions of the nose, face and skull, like most surgical procedures, is not new. It was utilized rather extensively a generation ago and was discarded because of the belief that the grafts either suppurated or were absorbed and replaced by fibrous tissue. Recently there has been a revival of the method. J. B. Brown,[1] Clair Straith, C. R. Straatsma and I have used cartilage preserved in alcohol to repair saddle nose and defects of the skull.

Successful grafts examined by external palpation appear to retain their size and consistency up to periods of over a year. The advantage of this procedure is obvious, as fresh cartilage from cadavers is easily obtainable in almost any desired quantity and a supply of cartilage preserved in alcohol may be kept on hand in the hospital laboratory for use when needed. It goes without saying that the donor must be proved to be free from syphilis.

The disadvantage of the method lies in the uncertainty as to the ultimate fate of the transplant. Theoretically, one would suppose that fresh cartilage killed by immersion in alcohol for thirty days or more and buried beneath the skin would meet one of three fates:

1. Absorption of the cartilage and partial replacement with connective tissue.

2. Invasion of the implanted cartilage framework by fibroblasts from the surrounding connective tissue of the host, which would occupy the cell spaces in the cartilage and take over the function of the dead cartilage cells.

Read before the Nose and Throat Section of the Academy of Medicine of Northern New Jersey, April 12, 1937.

1. Unpublished data. J. B. Brown has used cartilage preserved in alcohol since 1928 and has found it retained permanently in most instances.

3. Retention of the cartilage graft as a tolerated dead foreign body.

The experimental work reported in this paper was performed to determine the fate of the cartilage implant and the nature of the reaction in the surrounding connective tissue of the host.

THE HISTOLOGY OF CARTILAGE

All connective tissues, including cartilage, are derived from mesenchyme, which is a thin mixture of cells arising from the middle germ layer. Hence, the common cellular component of all connective tissue, the fibroblast, is a descendant of the primitive mesenchymal cell.

One must bear in mind that cartilage, bone, tendon, areolar tissue, fat and the dermis of the skin are all connective tissues with the same type of cellular element, the fibroblast. These tissues differ in physical properties because of their different intercellular substance. Thus, bone is rigid owing to a calcified substance between its cells; cartilage is firm but relatively elastic owing to a gelatinous intercellular material, and tendon is pliable and strong owing to the presence of tough bundles of fibers between its cells. Fat also is a connective tissue, but it has a contrasting predominance of cellular elements and a relatively small amount of intercellular material.

It is interesting to note that all types of connective tissue may be successfully transplanted as free autografts and tend to retain their original structure through complete survival, as with cartilage, or through absorption and partial replacement, as with bone and fat grafts.

Three chief varieties of cartilage are distinguished. In one, which is termed hyaline, the matrix, or intercellular substance, is almost clear and free from obvious fibers; in the other two, which are termed fibrocartilage, the matrix is pervaded by connective tissue fibers. When these are of the white variety, the tissue is white fibrocartilage; when they are elastic fibers, it is yellow, or elastic, fibrocartilage.

The costal and septal cartilages are of the hyaline variety and are surrounded by a fibrous membrane, the perichondrium. The cartilage itself consists of an intercellular matrix studded with small chambers, called lacunae, which contain the cartilage cells. The matrix of hyaline cartilage appears homogeneous and is composed of the special proteins—(1) collagen (on boiling it forms gelatin), (2) chondromucoid and (3) chondro-albuminoid.

The cartilage cells occur in groups of two, four, eight, etc., owing to the fact that these groups have originated from the division of a single cell, first into two, each of these again into two and so on. During life the protoplasm of the cell entirely fills the lacuna which it occupies in the matrix, but after death and as a result of fixation

the cells appear shrunken and often lose all trace of cell structure.[2] The more newly formed areas of matrix directly about the cartilage cells take on a deeper stain than the rest of the matrix, thus forming a framework, or capsule, about the cells.

Growth takes place from the deep layer of connective tissue cells of the perichondrium, which form a matrix substance about themselves separate from the perichondrium and become cartilage cells. Growth also occurs by division of cartilage cells, followed by the production of matrix about each cell separating them one from the other.

Cartilage in the adult is avascular. Large masses, such as the costal cartilages, may be invaded by an occasional blood vessel, apparently because the mass of tissue is so great that an adequate food supply by diffusion is impossible. Fluids from vessels in the perichondrium percolate through the matrix to the cartilage cells, and waste is eliminated by diffusion in the opposite direction.

In summarizing, it is seen that the matrix, or intercellular substance, of cartilage gives this structure its property of elastic rigidity and adapts it for its special function of support. This matrix is inanimate material, which is durable and is produced by activity of the living cartilage cell.

The cartilage cells locked away in the matrix are the living elements of cartilage, and while they are immobile they are active in the maintenance of their surroundings.

PREVIOUS EXPERIMENTAL WORK WITH LIVING CARTILAGE IN ANIMALS

The literature on cartilage over the past eighty years is so extensive that I have attempted to summarize only the most important work and to indicate its significance.

Paul Bert [3] was probably the first to transplant cartilage. From his experimental studies with animals he came to the conclusion that a cartilage graft retains its viability and leads to the formation of bone. (His work was probably done before 1865, which is the date of the first reference.)

Ollier,[4] in 1867, and Zahn,[5] in 1884, assumed a diametrically opposed view of the fate of adult animal cartilage transplants. They both noted

2. Sharpey-Schafer, E.: Essentials of Histology, ed. 13, Philadelphia, Lea & Febiger, 1934, p. 112.

3. Bert, P.: Sur la greffe animal, Compt. rend. Acad. d. sc. **61**:587, 1865.

4. Ollier, L.: Traité expérimental et clinique de la régénération des os et de la production artificielle du tissue osseux, Paris, V. Masson & fils, 1867.

5. Zahn, F. W.: Ueber das Schicksal der in den Organismus implantierten Gewebe, Virchows Arch. f. path. Anat. **95**:369, 1884.

degenerative changes in the grafts that led ultimately to their absorption. Zahn concluded that cartilage grafts degenerate, whether transplanted into the same animal (autografts) or into different animals (homografts and heterografts).

Fischer [6] was responsible for the origin of the theory that the survival of a cartilaginous graft is dependent on the presence of the perichondrium. In his experiments, costal cartilage degenerated after about eight weeks when transplanted as a homograft without its perichondrium. Cartilage transplanted with its perichondrium showed little alteration in structure over the same period.

I found expressed in this early experimental work on animals the three points of view as to the fate of adult cartilage transplants which still exist today.

1. Cartilage tends to survive when transplanted.

2. Cartilage tends to degenerate when transplanted.

3. Cartilage survives when transplanted with its perichondrium but tends to degenerate and disappear when transplanted without its perichondrium.

Illustrating the more recent work on cartilage transplants in animals, Leo Loeb,[7] in 1926, buried xiphoid cartilage with perichondrium and fat of a guinea-pig in the same guinea-pig (autograft) and in different guinea-pigs (homograft). He removed the cartilage at intervals of from one day up to five months and nineteen days. He found that after autotransplantation reaction in the tissues about the transplant was almost entirely lacking.

After homotransplantation, lymphocytes collected about the graft, and in places lymphocytes, connective tissue cells and blood vessels entered necrotic portions of the cartilage. The reaction to the graft began early, reached a maximum in about three weeks and from this time on ceased to a great extent. Both the autograft and the homograft remained up to five months and nineteen days.

Mannheim and Zypkin [8] performed fifty experiments with guinea-pigs, transplanting cartilage from one part of the animal to another part in the same animal. Their conclusions concerning sections examined up to one year after transplantation were:

1. The cartilage retained its specific structure in all cases.

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6. Fischer, E.: Ueber Transplantationen von organischem Material, Deutsche Ztschr. f. Chir. **17**:362, 1882.

7. Loeb, L.: Autotransplantation and Homoiotransplantation of Cartilage in Guinea-Pig, Am. J. Path. **2**:111, 1926.

8. Mannheim, A., and Zypkin, B.: Free Autoplastic Cartilage Transplantation, Arch. f. klin. Chir. **141**:688, 1926; abstr., J. A. M. A. **87**:2132 (Dec. 18) 1926.

2. Both degenerative and regenerative processes occurred.

3. The cartilage after transplantation into soft parts was better preserved than after transplantation into the skull.

4. Cartilage transplanted without its perichondrium was better preserved than cartilage transplanted with its perichondrium.

5. Free autotransplanted cartilage grafts form a good material for plastic repair.

Other important contributions were given by Tizzoni, Leopold, Seggel, Birch-Hirschfeld and Garten, Giani, Zoppi, Helferech and Enderlen, Rehn, Dalla Vedova, Galeazzi, Leo Loeb, Harbin and Moritz, Haas, Siebert, Saltykow, Solaro, Jentger and Allessandri.

PREVIOUS EXPERIMENTAL WORK WITH LIVING CARTILAGE IN MAN

König,[9] in 1896, was the first to use cartilage transplants in man. He buried segments of cartilage as wedges for the repair of partial destruction of the laryngeal and tracheal cartilages.

Von Mangoldt,[10] in 1899, successfully transplanted costal cartilage for support of the nose.

Nélaton and Ombredanne,[11] in 1904, buried costal cartilage in flaps on the forehead, which were later swung down to the nose.

Neuhoff,[12] in 1923, after an extensive survey of the literature concluded:

> Simple cartilage grafts, after transplantation, remain unaltered in appearance and in staining reaction for many weeks; only in grafts that are several months old does fibrillation of the cartilage begin. There is a gradual death of the cartilage cells, and they ultimately disappear. Vascularization of the graft, and replacement by fibrous tissue or calcification may occur, depending on the locality to which the graft is transplanted. The outstanding feature in the histologic fate of cartilage transplants is the long period of quiescence that precedes the final phase of degeneration and substitution.

In referring to transplants of rib cartilage Neuhoff stated:

> The cartilage is absorbed slowly, allowing adequate time for replacement by dense fibrous tissue that maintains the architecture of the graft.

He further said that remnants of cartilage have been found as long as two years after transplantation.

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9. König, F.: Reaction of Cartilage to Injury, Arch. f. klin. Chir. **124**:1, 1923; abstr., J. A. M. A. **81**:1646 (Nov. 10) 1923.

10. Von Mangoldt: Arch. f. klin. Chir. **39**:926, 1889.

11. Nélaton, C., and Ombredanne, L.: La rhinoplastie, Paris, G. Steinheil, 1904.

12. Neuhoff, Harold, and Hirschfeld, S.: The Transplantation of Tissues, New York, D. Appleton and Company, 1923, pp. 205-215.

In opposition to this point of view the earlier experimental work of Staige Davis,[13] in 1917, gave clinical evidence of the permanence of rib cartilage transplanted in the nose.

Gillies[14] also, in 1920, stated that no changes other than curvature were found in any of his successful autocartilage grafts and in only a few of the homografts was the cartilage replaced by fibrous tissue as a late sequel. Three years was the longest time that Gillies had a graft under observation.

Gillies also buried an autograft and a fresh homograft of cartilage beneath the abdominal skin of the same patient. He removed both grafts after eighteen months and on section found the cartilage alive and active in both cases. The cells in the homograft were more vacuolated and showed more degenerative changes than did those of the autograft. The autograft appeared as normal adult cartilage.

Other important contributions were given by Henle, Lexer, Eiselsberg, Sgambati, Schmieden, Tuffier, Morestin, Gosset, Leriche, Queim and Bossi.

SUMMARY OF TRANSPLANTATION OF LIVING CARTILAGE IN MAN

The weight of more recent evidence appears to favor the view of Gillies and Davis that autotransplants of cartilage, with or without their perichondrium, tend to survive as living cartilage in man.

Homotransplants of cartilage appear to survive in some cases and in others to undergo a slow degenerative change.

PREVIOUS EXPERIMENTAL WORK WITH THE TRANSPLANTATION OF DEAD CARTILAGE

Even in the literature of over fifty years ago there were reports of the homotransplantation of cartilage which was dead or fixed with the use of alcohol, heat or other agents.

Prudden,[15] in 1881, transplanted cartilage grafts killed by immersion in 95 per cent alcohol. His results showed degeneration and partial absorption of all these grafts.

Nageotte,[16] in 1922, buried rabbit cartilage fixed in alcohol in rabbits' ears. He found that the fixed cartilage lost its basophilic staining and

13. Davis, J. S.: Some of the Problems of Plastic Surgery, Ann. Surg. **66**: 88, 1917.

14. Gillies, H. O.: Plastic Surgery of the Face, London, Oxford University Press, 1920, pp. 13-14.

15. Prudden, T. Mitchell: Experimental Studies on the Transplantation of Cartilage, Am. J. M. Sc. **82**:360, 1881.

16. Nageotte, J.: The Organization of Material in Its Relation to Life, Paris, Masson & Cie, 1922.

promoted a metaplasia of the fibroblasts which surrounded it into chondroblasts with the consequent new formation of cartilage and bone. Phagocytes and fibroblasts penetrated only into the open cavities at the periphery of the graft and in the cracks, which eventually gave access to the surrounding cells of the host tissue.

Polettini,[17] in 1922, pursued similar experiments with cartilage fixed in alcohol. His sections showed penetration of fibroblasts and formation of connective tissue fibers within the cartilage where no cavities or cracks previously existed as a point of entry. He also found cartilage and bone formation in the vicinity of the graft.

Nigrisoli,[18] in 1927, transplanted calves' cartilage fixed in 95 per cent alcohol into the kidney and bone of guinea-pigs. He removed his grafts in from six to one hundred and eighty days and found that the bulk of the graft survived in each case. Connective tissue from the host tended to invade the cartilage, and calcifications were found in the grafts. Rarely did he find new-formed bone and cartilage outside the graft.

Didier and Guyon,[19] in 1928, transplanted killed cartilage grafts in the ear, shoulder and linea alba of rabbits. They observed grafts up to eight and one-half months and found calcification and invasion by fibroblasts from the host tissue. One subject showed ecchondroses, and another showed ossification, with the formation of fatty marrow. Two showed a clear histologic picture of bone formation.

The newer literature, while reporting the survival of transplants of killed cartilage, emphasizes the reaction of the host tissue and a gradual modification of the cartilage. The modification includes calcification and ossification as progressive changes and invasion by fibroblasts with degeneration of the matrix as degenerative changes.

These changes do not interfere with the survival of the block of cartilage up to periods as long as one year. The work with fixed or dead cartilage transplants summarized was all performed on animals.

EXPERIMENTAL PROCEDURE

All this work was performed on human beings. The experimental subjects, as in the previous work with dermal grafts,[20] were patients who underwent rib graft

17. Polettini, B.: Experimental Growth of Bone and Cartilage Grafts, Arch. ital. di chir. **6**:179, 1922-1923.

18. Nigrisoli, P.: Experimenti di innesto di cartilagine fissata nel rene e di sostituzione di parti scheletriche con cartilagine fissata, Arch. per le sc. med. **49**: 689, 1927.

19. Didier, R., and Guyon, L.: Production de cartilage et d'os, au sein de greffes vivantes et mortes, chez le lapin, Compt. rend. Soc. de biol. **98**:443 (Feb. 17) 1928.

20. Peer, L. A., and Paddock, R.: Histologic Studies on the Fate of Deeply Implanted Dermal Grafts, Arch. Surg. **34**:268 (Feb.) 1937.

operations for the repair of saddle nose. In these operations it is customary to hoard excess rib cartilage beneath the skin of the chest for use in case the cartilage inserted in the nose fails to remain in place. When the nasal repair is satisfactory, the excess cartilage beneath the skin on the chest is removed and discarded.

Segments of human costal and septal cartilage preserved in alcohol were inserted beneath the skin of the chest as homografts together with the hoarded rib cartilage and specimens were removed for examination at intervals of seven days, eight days, thirty-two days, four months and fourteen months.

DESCRIPTION OF SECTIONS

The sections described here were prepared by Mr. David J. McKinnon of the Newark Eye and Ear Infirmary. Dr. Royce Paddock aided in reviewing the foreign literature.

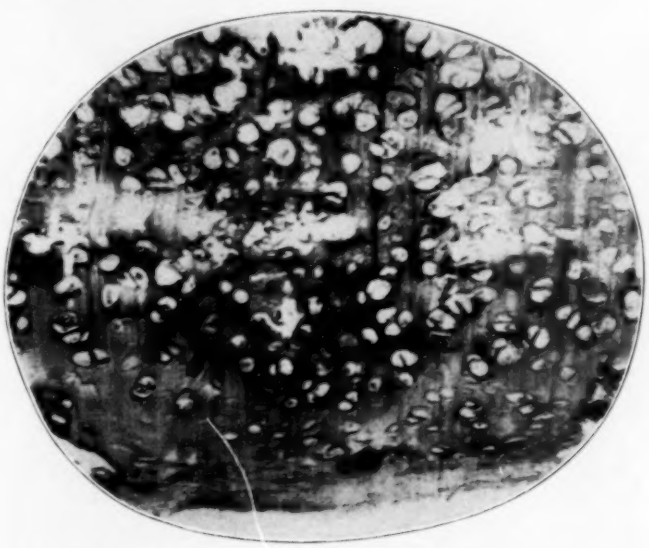

Fig. 1.—Control. The section shows a segment of septal cartilage preserved in alcohol for one month, sectioned and stained with hematoxylin and eosin. The matrix is stained irregularly, with darker areas about the lacunae. The cartilage cells are shrunken owing to fixation.

The sections represent segments of human costal and septal cartilage preserved in 50 per cent alcohol for one month or more and transplanted beneath the skin of the chest of other human subjects. At intervals of seven days, eight days, thirty-two days, sixty-two days, four months and fourteen months an implant was excised together with the surrounding connective tissue of the host and placed in Zenker's solution. After an implant was sectioned in the usual manner, the tissues were stained with hematoxylin and eosin and with Mallory's connective tissue stain. Photographs were taken under high power and low power magnification.

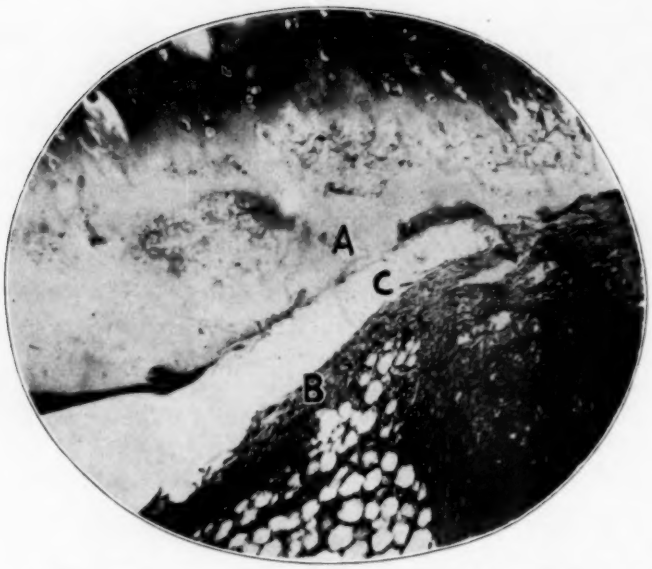

Fig. 2.—Hematoxylin and eosin stain. Segment of rib cartilage buried for seven days. *A* represents the cartilage; *B*, the capsule of connective tissue which has formed about the cartilage, and *C*, an artificial space due to cutting of the section.

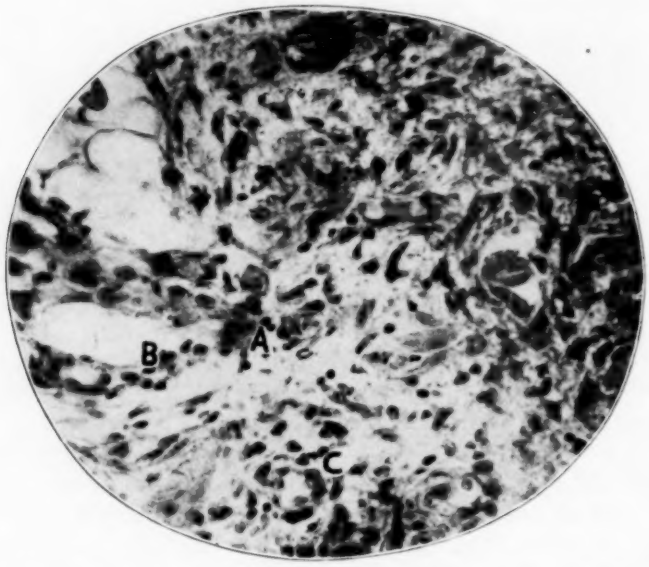

Fig. 3.—Hematoxylin and eosin stain. A high magnification photograph showing the reaction in the host tissue surrounding the cartilage transplant after seven days. *A* represents a group of polymorphonuclear leukocytes; *B*, a collection of large mononuclear cells and *C*, a cluster of multinucleated cells, which are probably beginning giant cells. There is also proliferation of fibroblasts and dilatation of blood vessels.

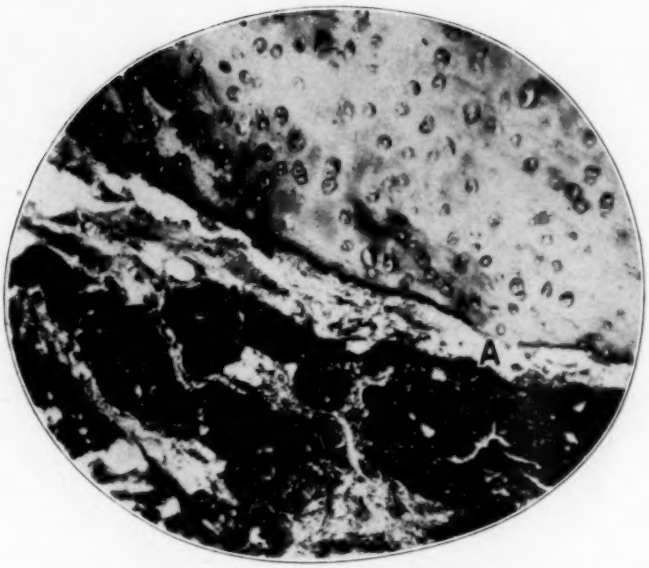

Fig. 4.—Mallory's connective tissue stain. Section of septal cartilage buried eight days. A capsule of dense connective tissue has formed about the graft *A*. The space between the cartilage and the capsule of connective tissue is occupied by broken-down blood cells. The cartilage shows no evidence of invasion from the host tissue and appears well preserved.

Fig. 5.—Mallory's connective tissue stain. Section of septal cartilage buried thirty-two days. A capsule of dark-staining connective tissue is in close contact with each border of the cartilage *A* and *B*, and the cartilage is well preserved, with no evidence of degeneration or invasion.

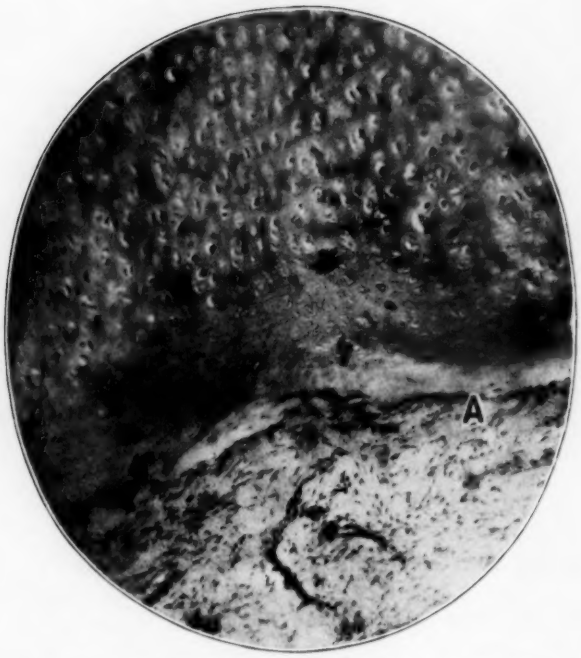

Fig. 6.—Hematoxylin and eosin stain. A section of rib cartilage buried for sixty-two days. *A* represents the capsule of dense connective tissue in close contact with the cartilage. No degeneration or invasion of the cartilage was found in any of the sections buried for sixty-two days.

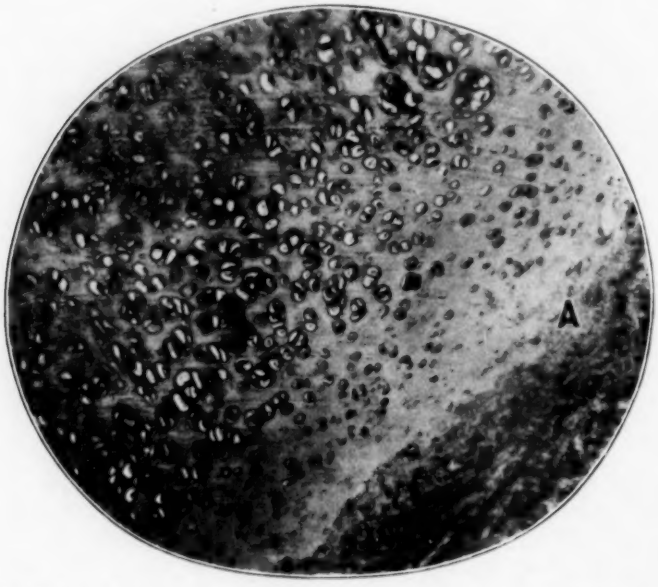

Fig. 7.—Mallory's connective tissue stain. A higher magnification photograph of rib cartilage buried for sixty-two days. The capsule of connective tissue is indicated by *A*.

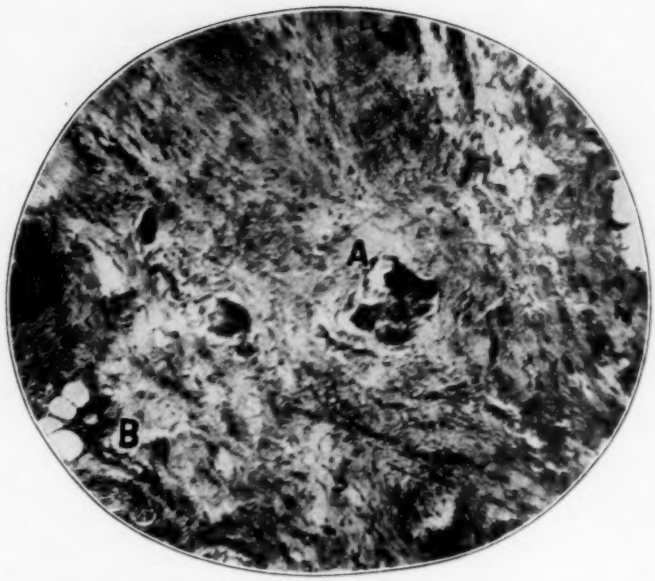

Fig. 8.—Hematoxylin and eosin stain. The section shows an area of the host tissue surrounding a cartilage transplant buried for sixty-two days. Foreign body giant cells are seen at *A* and *B*, but otherwise there is a rather general absence of reaction in the host tissue.

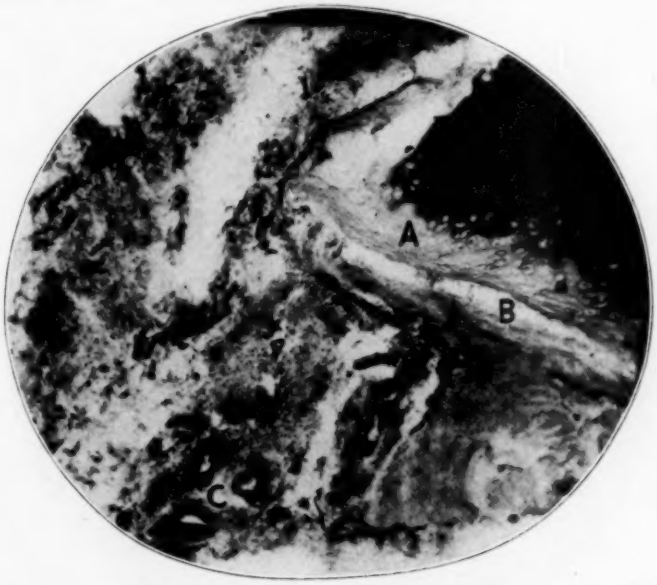

Fig. 9.—Hematoxylin and eosin stain. A section of rib cartilage buried for four months, with the adjacent host tissue. *A* represents cartilage; *B*, the capsule of connective tissue, and *C*, a group of thick-walled blood vessels in the connective tissue of the host. The cartilage showed no evidence of degeneration or absorption in any of the sections buried for four months.

Fig. 10.—Mallory's connective tissue stain. Section of septal cartilage buried for fourteen months. One end of the cartilage is shown, with the adjacent connective tissue of the host. The connective tissue of the host is penetrating the cartilage at *A*, separating it into two portions, *B* and *C*. The free margins of the cartilage show a moth-eaten appearance where absorption has taken place. The large dark-staining bodies are giant cells.

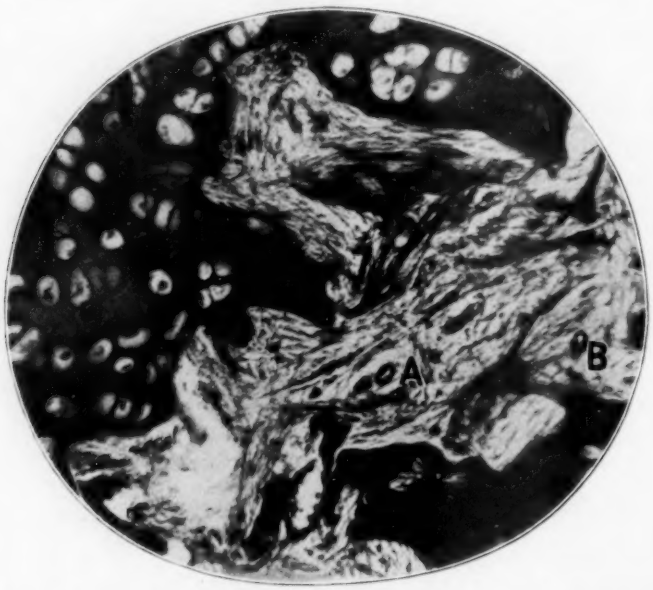

Fig. 11.—Mallory's connective tissue stain. A higher magnification at fourteen months, showing the fibers of the invading connective tissue of the host, with blood vessels at *A* and *B*. The free margins of the cartilage show erosion.

Fig. 12.—Hematoxylin and eosin stain. Section of septal cartilage buried for fourteen months, showing a large calcified area. Two large giant cells are indicated by *A* and *B*, and at *C* there is a chain of large mononuclear cells which resemble the osteoblasts of normal bone.

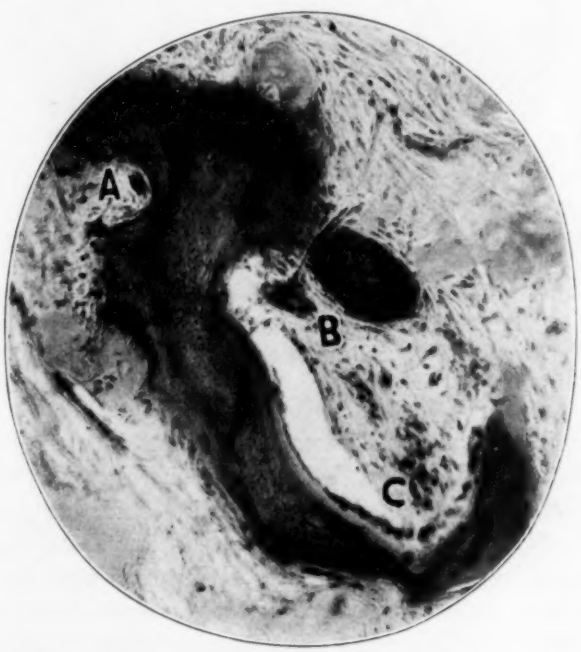

Fig. 13.—Hematoxylin and eosin stain. Medium power magnification of a calcified area in a cartilage transplant buried for fourteen months. The two large giant cells are indicated by *A* and *B*. The chain of large mononuclear cells resembling osteoblasts is shown at *C*.

The most striking feature in the sections was the absence of invasion of the cartilage transplant by the host tissues up to and including the specimen buried for four months. Some time between the fourth and the fourteenth month the host tissues became aggressive, and in the sections buried for fourteen months definite invasion of the cartilage was found. Another important observation was the early formation of a capsule of connective tissue about the transplanted cartilage. This was found in all the sections buried for seven or eight days and persisted in all the later sections.

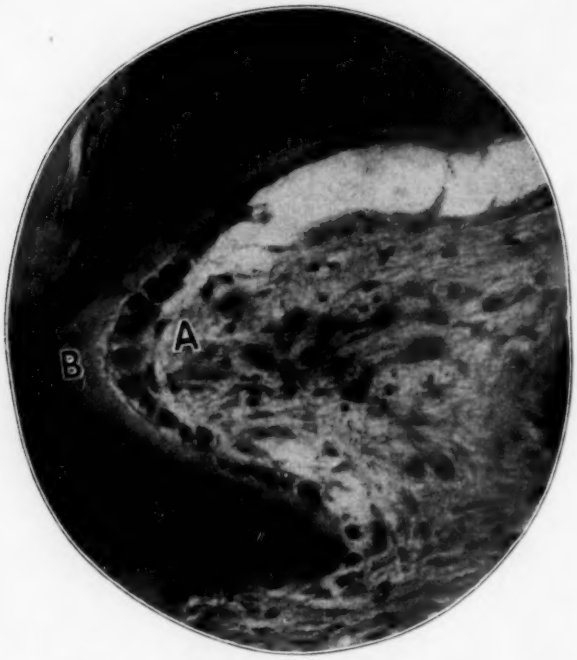

Fig. 14.—Hematoxylin and eosin stain. High power magnification of a calcified area in a cartilage transplant buried fourteen months. *A* indicates a chain of large mononuclear cells resembling osteoblasts. *B* indicates two vital-appearing cells embedded in the calcified area.

Reaction in the connective tissue of the host about the transplanted cartilage in the sections buried for seven or eight days was of the foreign body type (fig. 3). This consisted of a rather general infiltration with polymorphonuclear leukocytes, large and small mononuclear cells and beginning giant cell formation. There was also a proliferation of the fibroblasts and engorgement of blood vessels in the host tissue about the cartilage. This reaction was present to a less extent in the

sections buried for thirty-two days but was absent in those buried for sixty-two days, except for the presence of occasional large foreign body giant cells (fig. 8).

From thirty-two days to four months, inclusive, the transplanted cartilage remained in place as a tolerated dead foreign body, subjected to no invasion from the surrounding host tissues. Some time between the fourth and the fourteenth month the host tissues became active, and in the sections buried for fourteen months fibroblasts were seen growing into each end of the cartilage, splitting it into two separate masses. New blood vessels were seen in the invading connective tissue, and giant cells were scattered at intervals near the eroded edges of the

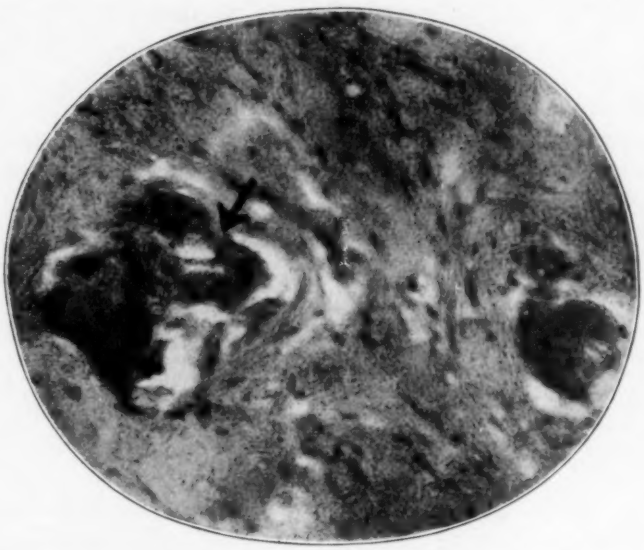

Fig. 15.—Hematoxylin and eosin stain. Section photographed under polarized light. The section shows two large giant cells in the connective tissue of the host surrounding the cartilage transplant at fourteen months. The larger of the giant cells contains two crystalline substances which may represent degenerative products of ingested cartilage.

cartilage. At about the center of the cartilage in the sections buried for fourteen months a rather large hook-shaped area of calcification was seen. This, I believe, represented a calcification of preexisting cartilage, since lacunae containing shrunken cartilage cells were demonstrated in the mass of calcified substance. Along one border of this calcified area was a chainlike formation of large mononuclear cells, which resembled the osteoblasts of true bone. In support of this conception was the presence of large vital-appearing cells embedded within

the calcified substance (fig. 15). If these cells were to remain as living cells, channels or canaliculi would have to develop to provide for their nourishment, and one may consider the calcified area as living bone.

SUMMARY

Sections of human costal and septal cartilage preserved in 50 per cent alcohol were transplanted beneath the skin on the chest of other human beings and removed at intervals of from seven days to fourteen months.

A reaction of the foreign body type was seen in the host tissues about the transplants. This reaction lasted until the thirty-second day period and was largely absent in the sections removed later.

From thirty-two days to and including four months the grafts remained as tolerated dead foreign bodies.

In the sections buried for fourteen months fibroblasts from the host tissue grew into the cartilage graft, splitting it into two separate masses. There was definite absorption of the cartilage, and in one area calcification of the cartilage.

One cannot advocate or discourage the clinical use of pickled cartilage grafts on the basis of these experiments. If absorption of the cartilage and replacement by fibrous tissue and new bone maintain the contour of the grafts, they may constitute a desirable filling substance. If, on the other hand, the architecture of the grafts is not retained, then they do not possess the qualities desirable for a good filling substance. I hope to report in a later paper on sections buried up to two and one-half years.

EFFECT OF LIMITED COCHLEAR LESIONS ON COCHLEAR POTENTIALS AND MIDDLE EAR MUSCLE REFLEXES

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CHICAGO

The discovery of the Wever and Bray phenomenon in 1930 provided the stimulus for a great amount of experimental work in the field of audition. The discovery that electrical potentials can be picked up from the cochlea, auditory nerve and auditory tracts from which sounds entering the animal's ear can be reproduced furnished a promising method for the investigation of auditory function in the animal.

At the same time other investigators have continued making observations, using various types of conditioned reflexes as tests of hearing.

Still other investigators have studied the reflex contractions of the muscles of the middle ear in response to sound and have shown that these contractions may be used as an indicator of hearing. Observations of the reflex of the stapedius muscle in man have permitted the correlation between the reflex and the sensation of hearing.

Various observations have been made with the aforementioned types of indicators of hearing under both normal and pathologic conditions of the ear. Under some conditions, the results obtained have corresponded closely, while under other conditions, the different methods used for testing the hearing function have given widely divergent results.

The variation in results has indicated the necessity of making further studies whereby the different methods of testing cochlear function may be compared both under normal and under specific pathologic conditions.

In the following experiments a comparative study has been made of the effects of perforating lesions of the cochlea on the electrical potentials picked up from the cochlea and on the acoustic reflex response of the muscles of the middle ear.

Work done in part under a grant of the Douglas Smith Foundation.

Department of Surgery and Department of Physiology, University of Chicago.

METHOD

Our procedure consisted in determining the threshold of the reflex of the acoustic tensor tympani muscle and the electrical response of the cochlea in the rabbit. The animals were tested by both methods, first under normal conditions and then after a perforating lesion of the cochlea had been made. Light anesthesia with ethyl carbamate (urethane) was used. The bulla ossea was exposed and opened from the ventral side. By this method no interference with any part of the ossicular chain or the tympanic membrane is necessary. The production of tone was provided by a 2-A audiometer connected with an amplifier and a loud-speaker. The thresholds were determined by direct observation, a small lens being used. The lowest intensity of a tone necessary to give a visible contraction is taken as a threshold reading. The threshold values for different pitches are plotted as the threshold curve.

The electrical potentials were led off the cochlea by a bipolar Adrian electrode, which usually was placed in the niche of the round window, and were conducted by means of a shielded cable to the amplifier, from which they were conducted to a loud-speaker in a third room. For a number of experiments a cathode ray oscillograph was used. Control tests proved that by this arrangement microphonic action of the amplifier was avoided. Furthermore, the response was specifically cochlear, since it was not obtainable except when the electrode was placed on the cochlea. While the electrical potentials were being determined, the acoustic stimuli were furnished by means of tuning forks, the human voice and whistling.

FUNCTIONAL TESTS

The left bulla ossea was opened first, and the left labyrinth was destroyed. After this the right side was opened and the threshold of the contractions of the tensor tympani muscle was determined. The electrode was then placed in the niche of the round window, and the presence of a normal electrical response was demonstrated. The observer at the loud-speaker was able to repeat the numbers and words which were spoken into the ear of the rabbit. A rough quantitative measurement was made by increasing the distance between the animal and the source of sound until the threshold value was determined.

After the tensor reflex and the cochlear response were investigated under normal conditions, a small perforation was made in the promontorium by means of a fine needle. As soon as the needle perforated the promontorium, labyrinthine fluid escaped and filled the bulla. With few exceptions, after the hole had been made the acoustic response of the tensor tympani muscle rapidly decreased and was absent within a few minutes (fig. 1). The electrode was replaced in the niche of the round window, and in nearly all cases an undiminished electrical response could be demonstrated in the loud-speaker. In several cases the electrical response was even better than before. The vibrations of a tuning fork of 100 double vibrations were clearly visualized on the oscillograph in spite of the fact that the apical part of the cochlea had

been destroyed. The sound from higher-pitched tuning forks as well as the spoken voice was also clearly demonstrated on the oscillograph.

Another series of animals were operated on under sterile precautions and tested several days later. In these cases the perforation was closed by the beginning healing process so that there was no longer any escape of labyrinthine fluid. The results of the experiments with nonacute lesions were not uniform, at least not so far as the reflex response of the tensor tympani muscle was concerned. A number of animals still showed acoustic contraction but always to a diminished degree (fig. 2). In another group no reflex contraction could be elicited in response to sound, either by air conduction or by bone conduction.

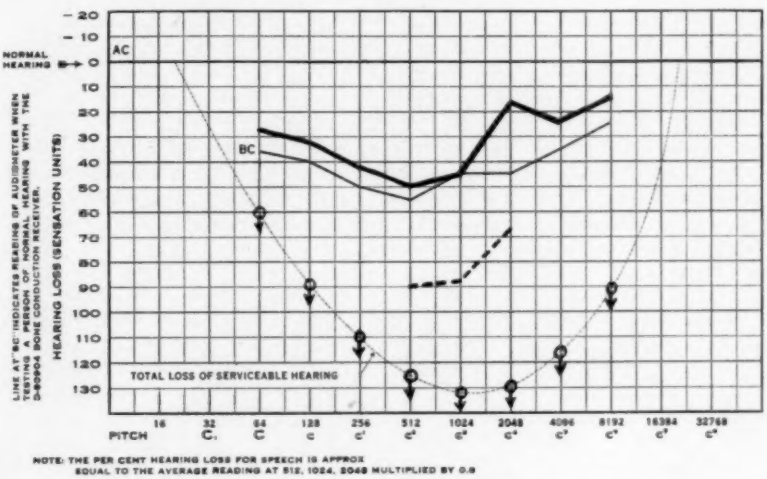
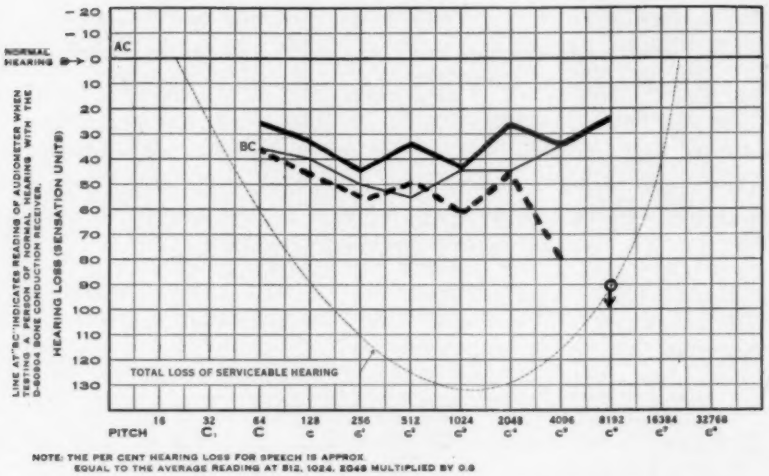

NORMAL HEARING
AC
BC
HEARING LOSS (SENSATION UNITS)
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
NOTE: THE PER CENT HEARING LOSS FOR SPEECH IS APPROX EQUAL TO THE AVERAGE READING AT 512, 1024, 2048 MULTIPLIED BY 0.8

Fig. 1.—Audiogram of a rabbit under anesthesia with ethyl carbamate (urethane). The animal had an acute limited cochlear lesion. The apical coil and half of the middle coil had been destroyed. The solid line indicates the normal threshold of the reflex of the tensor tympani muscle for air-conducted tones; the broken line represents the bone conduction. There was complete loss of the acoustic muscle reflex for both air-conducted and bone-conducted sounds. The cochlear potentials were unchanged. The histologic appearance of the cochlea is shown in figure 4.

In all the animals the electrical response was obtained and appeared to be of undiminished intensity.

In several of the experiments in which the perforation was small the tensor threshold was determined every three minutes after the hole was drilled. Figure 3 shows the gradual loss of the acoustic reflex in one case.


NORMAL HEARING
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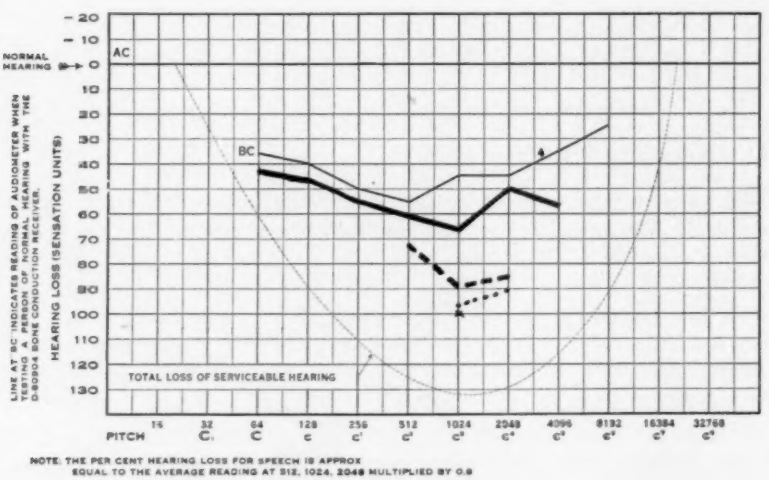
HEARING LOSS (SENSATION UNITS)
PITCH
NOTE: THE PER CENT HEARING LOSS FOR SPEECH IS APPROX. EQUAL TO THE AVERAGE READING AT 512, 1024, 2048 MULTIPLIED BY 0.8

Fig. 2.—Functional test of the cochlea of a rabbit which was operated on eleven days previously. A hole was drilled into the right apical coil. The functional test was made with the animal under anesthesia with ethyl carbamate. The solid line indicates the normal reflex threshold of the left intact labyrinth. The broken line represents the reflex function of the right cochlea after destruction of the left inner ear.


NORMAL HEARING
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TOTAL LOSS OF SERVICEABLE HEARING
HEARING LOSS (SENSATION UNITS)
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NOTE: THE PER CENT HEARING LOSS FOR SPEECH IS APPROX. EQUAL TO THE AVERAGE READING AT 512, 1024, 2048 MULTIPLIED BY 0.8

Fig. 3.—Audiogram of a rabbit under anesthesia with ethyl carbamate. A series of curves was taken at three minute intervals after a fine hole was made in the apical coil of the right cochlea. The solid line represents the normal threshold of the reflex of the tensor tympani muscle. The broken line indicates the threshold three minutes later and the dotted line, six minutes later. The cross represents the only remaining acoustic response after nine minutes. Thereafter, no reflex could be elicited.

COMMENT

The widely divergent results obtained by various investigators with the use of different methods for determining the hearing in the experimental animal prompted this group of experiments, in which two indicators of hearing were used in the same experiment on the same animal. Dworkin [1] had found that he could make conditioned cats deaf by drilling a small hole in the cochlea. On the other hand, Bast and Eyster [2] found that destruction of as much as the upper three coils of

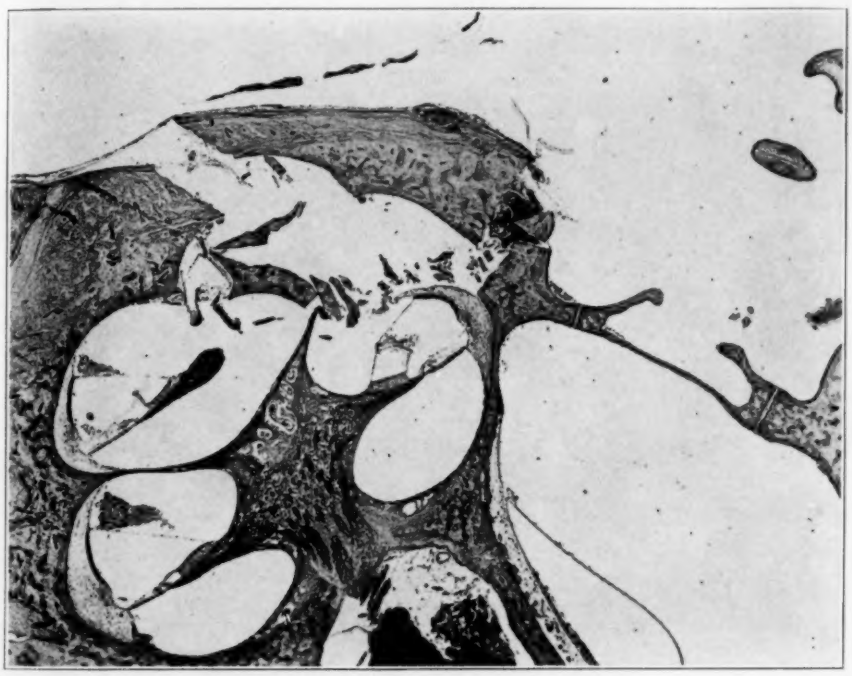

Fig. 4.—The cochlea of a rabbit after acute destruction of the apical half of the cochlea. A functional test revealed disappearance of the muscle reflex of the middle ear. The cochlear potentials led off the niche of the round window were grossly unchanged.

a guinea pig's cochlea did not markedly impair the cochlear response. Since these experiments were carried out, further comparative studies have been made by several groups of investigators (Davis, Lurie and Dworkin,[3] Culler [4] and Fowler and Forbes [5]).

1. Dworkin, S.: Ann. Otol., Rhin. & Laryng. **44**:803, 1935.
2. Bast, T. H., and Eyster, J.: Ann. Otol., Rhin. & Laryng. **44**:792, 1935.
3. Davis, H.; Lurie, M. H., and Dworkin, S.: Tr. Am. Otol. Soc., to be published.
4. Culler, E. A.: Tr. Am. Otol. Soc., to be published.
5. Fowler, E. P., Jr., and Forbes, T. W.: Am. J. Physiol. **117**:24, 1936.

In our experiments the muscle reflexes of the middle ear and the electrical response of the cochlea were tested in both acute and chronic conditions.

The acute disturbance with the loss of labyrinthine fluid produced little change in the cochlear response but abolished the reflex.

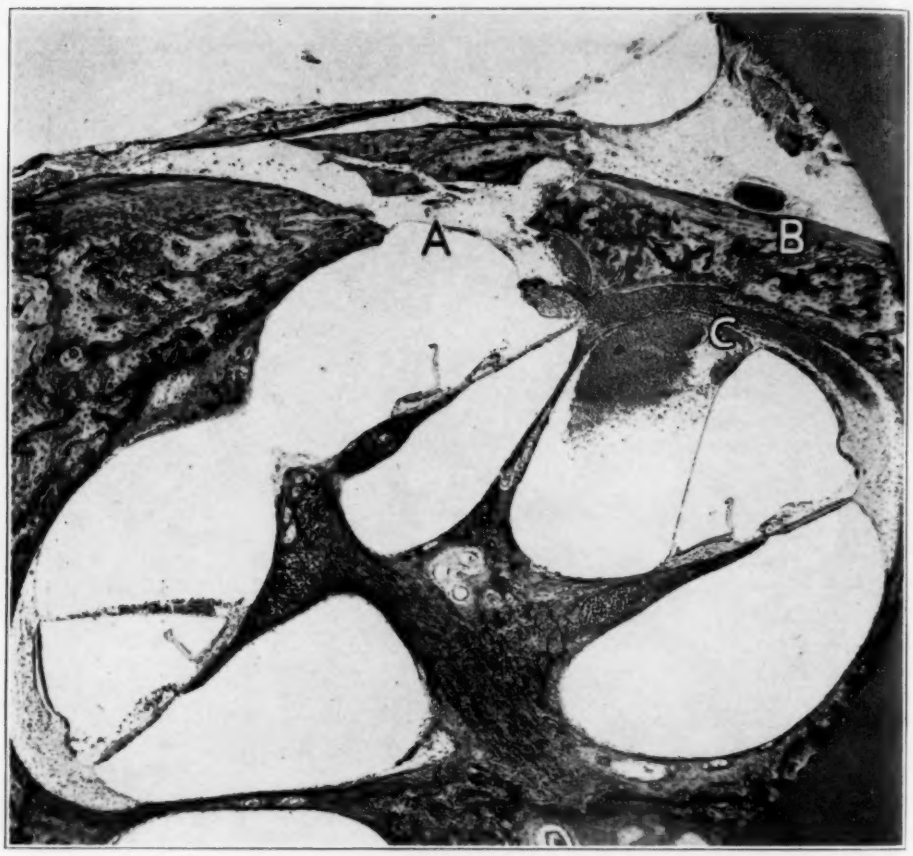

Fig. 5.—The cochlea of a rabbit. A drill hole was made in the promontorium two days previous to the functional test. At *A* the perforation is sealed with fibrin; at *B* a fracture in the promontorium is filled with blood, and at *C* hemorrhage into the spiral ligament and into the scala vestibuli is present. No reflex response of the tensor tympani muscle was obtainable. The electrical cochlear potentials were grossly unchanged.

The loss of the reflex response seems to be closely associated with the loss of labyrinthine fluid, as could be observed in the case of the small lesion, in which the loss of fluid was slow. On the other hand, even in the large lesions the cochlear response remained grossly

unchanged. The independence of the cochlear response from the hydrostatic conditions within the cochlea is clearly demonstrated by these experiments and has been previously demonstrated by Bast and Eyster in the case of the guinea pig's cochlea.

This finding is compatible with the hypothesis of Lurie and Davis, who expressed the belief that the electrical potentials originate in and

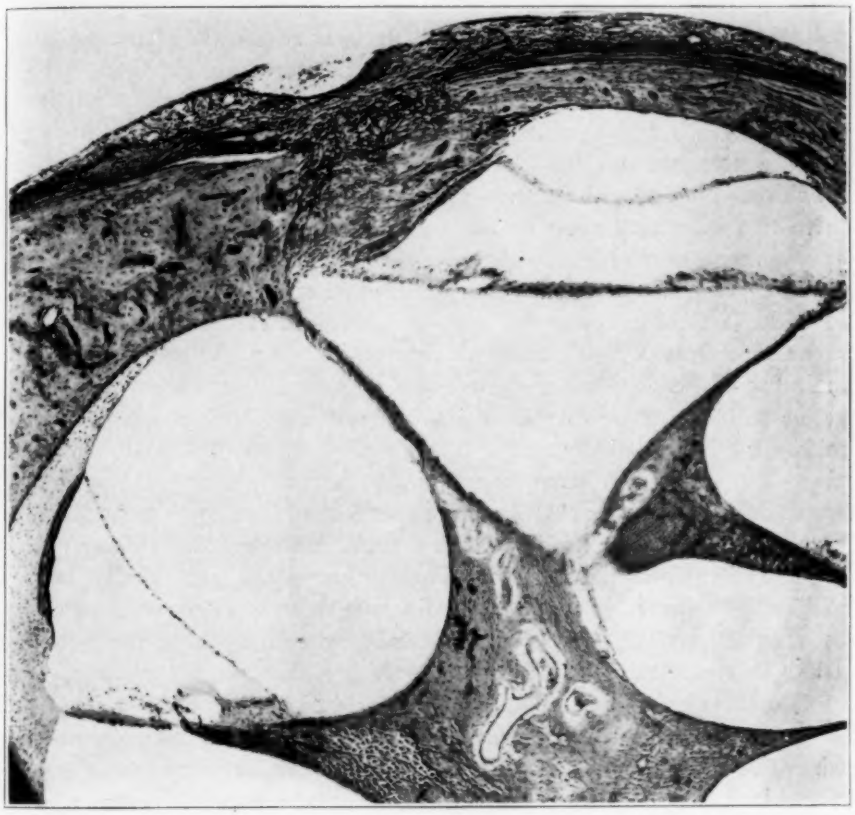

Fig. 6.—The cochlea of a rabbit. A fine hole was drilled into the apical coil eleven days previous to the functional test. The hole has been closed by connective tissue and newly formed bone. The result of the functional test is given in figure 2.

are dependent on the hair cells. Lurie could demonstrate that an estimation of the expected electrical activity of the cochlea can be made by the histologic appearance of the hair cell alone. The acute perforating lesion of the cochlea may therefore be a condition in which the hair cells and the electrical potentials are comparatively little disturbed. On the other hand, the acoustic reflex of the muscles of the middle ear

is incompatible with such severe changes of hydrostatic conditions as occurred in the experiment with acute lesions.

In the experiment with chronic lesions the healing of the perforation allows some restoration of equilibrium within the cochlea, and therefore the discrepancy between the two methods of estimating function is less marked.

It should be mentioned that the limen for the threshold of hearing is lower than that for the muscle reflex; however, the experiment revealed a functional loss of over 70 decibels as shown by the reflex, while the electrical response remained grossly unchanged.

These experiments were designed to throw light on the reliability of our methods employed as indicators of the cochlear function in the experiment with animals. The results show clearly that under the conditions that prevail in the experiments with acute lesions one of these two indicators must be at fault. There is no question that the development of cochlear potentials must be considered as a normal biologic phenomenon present in each normal cochlea. But as long as so little is known about the role of the electrical potentials in the process of hearing, one must be careful in using the method as an indicator of hearing under varied and uncontrolled conditions. Compared with the electrical potentials, the reflex of the muscles of the middle ear is a relatively simple biologic phenomenon, the anatomy and physiology of which have been known for many decades. Its use as an indicator of hearing in the experimental animal was proposed long after the reflex itself was thoroughly investigated. Finally, the relation of the muscle reflex of the middle ear to the sensation of hearing has been carefully studied in the human being. Many comparisons between the stapedius reflex and the hearing both by bone conduction and by air conduction have been made.[6]

The investigation of limited cochlear lesions is one example in which a difference in the result of functional tests has been observed when two methods for determining cochlear function were used.

It is hoped that comparative studies of the present methods for studying cochlear function will be made whenever possible. Such studies not only will serve to evaluate our experimental methods but will give a better understanding of the mechanism of hearing.

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6. Lindsay, J. R.; Kobrak, H., and Perlman, H. B.: Relation of the Stapedius Reflex to Hearing Sensation in Man, Arch. Otolaryng. **23**:671 (June) 1936.

INFECTION OF THE BLOOD STREAM

MEDICAL TREATMENT, WITH SPECIAL REFERENCE TO TRANSFUSIONS OF IMMUNIZED BLOOD; REPORT OF CASES

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During the period immediately prior to and following the World War, considerable emphasis was given to the surgical treatment of infection of the blood stream complicating acute and chronic mastoiditis. Little attention was given to the medical therapy. The modern trend, however, is in the direction of a more efficient application of various medical procedures.

The nearest approach to the ideal medical treatment at this time is the transfusion of whole immunized blood from compatible convalescent donors. A careful study of the blood is essential to the successful management of this condition. The knowledge of the nature of the initial infection determines to some degree the possibility of septicemia's developing and to some extent the type of treatment necessary. Cultures of the blood, when properly made, constitute one of the most valuable laboratory aids, but a positive or negative culture should not be the deciding factor in the management of any patient with septicemia. The otologist should be a clinician first and last, and in instituting treatment of any kind, medical or surgical, the well-being of the patient is the most important factor, regardless of laboratory findings.

There are many different views of the medical treatment, as shown by the contributions in the literature relative to this subject, but all agree that the primary focus of infection must be adequately treated surgically. While medical therapy has received emphasis in recent years, no one has been bold enough to advocate supplanting surgical drainage.

The transfusion of human blood is one of the advances of modern medicine. The patient receives on demand that which he cannot supply for himself. Transfusions were formerly used, except in rare instances, as a measure of last resort, but with an improved technic for determining the type of blood of donors and a better understanding of their selection,

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Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Eighth Annual Session of the American Medical Association, Atlantic City, N. J., June 10, 1937.

this mode of therapy is no longer considered dramatic, nor is its use confined to fatal stages of illness. Our early conception of blood transfusion was that it acted only as a supportive measure, but further clinical experience reveals that the patient receives not only hemoglobin and red blood cells but, still more important, fresh leukocytes for the elaboration of bactericidal substance, complement, opsonin, agglutinins, leukins and plakins. If this mode of therapy is to attain the place in the treatment of infection of the blood stream which it rightly deserves, the selection of the donor must be given specific consideration. The selection at random of a compatible donor is no longer justifiable. Modern science and thought call for the selection of a specifically compatible donor who (1) is convalescent, (2) possesses specific opsonizing antibodies, as shown by the opsonic index of his blood against the particular organism, (3) is nonspecifically immunized or (4) is specifically immunized.

While Streptococcus haemolyticus is frequently found in septicemia and may by serologic tests be divided into from ten to twenty strains, clinical studies suggest that the serum and blood from patients convalescent from various streptococcic infections frequently contain an immune substance antagonistic to similar but not necessarily identical organisms. This clinical observation suggests that transfusions of whole blood from compatible convalescent donors is at least a therapeutically specific method for the treatment of this infection. When possible, the selection of a young man as a compatible convalescent donor is preferable. In the selection of a donor the surgeon should follow the accepted principles of typing to avoid the error of using a donor whose blood is not of the patient's group and should bear in mind that every donor should be free from constitutional diseases, such as syphilis, malaria and asthma.

Jansky and Moss have classified human blood into groups, but in the interest of safety and simplicity, Kolmer and others recommended dropping the classifications of Moss and Jansky and employing only that of Landsteiner, which is as follows:

Landsteiner Group A,	corresponding to	II	of Moss and	II	of Jansky
Landsteiner Group B,	corresponding to	III	of Moss and	III	of Jansky
Landsteiner Group AB,	corresponding to	I	of Moss and	IV	of Jansky
Landsteiner Group O,	corresponding to	IV	of Moss and	I	of Jansky

The transfusions should be given at frequent intervals. The well-being of the patient determines the frequency of the transfusions. Small amounts of blood should be given at a time—not more than

150 cc. So long as there is definite clinical evidence of advancing sepsis, transfusions should be repeated.

Baum, of Denver, advocates the use of convalescent serum through the collection and storing of serum from persons convalescent from all types of streptococcic infection. He determines by agglutination the most specific serum for the particular infection. He has personally used this method in over fifteen hundred cases with good results. The principle is sound, and no doubt this method will be used more extensively in the future.

When compatible convalescent donors are not available, the next choice is a transfusion of nonspecifically immunized blood. This method has been advocated by Brody and Stephenson. This consists of giving the compatible donor intravenous injections of typhoid vaccine containing from 50,000,000 to 75,000,000 killed organisms and using the blood approximately seven hours later, after the acute reaction has subsided. The blood of the donor now carries fresh leukocytes of value and other antibacterial factors to the hard-pressed patient.

Hooker, Dick and others have advocated the use of specifically immunized blood prepared by repeated subcutaneous injections of the vaccine prepared from the organism infecting the patient. In cases of acute hemolytic septicemia there is not sufficient time for immunization of donors, but this method of preparation of the donor is advisable in staphylococcic septicemia, as the patient usually requires transfusions over a prolonged period.

Along with the timely administration of transfusions of immunized blood, the intelligent use of antistreptococcus serum is a valuable aid. The success of this mode of therapy depends on its early use and adequate dosage. As in the specific selection of a donor for a transfusion, one should by laboratory tests select antistreptococcus serums possessing agglutinating titer for the streptococcus recovered from cultures of the blood. Preference should be given to the serum of the highest titer. Before administering horse serum, proper tests should be made to determine the possibility of sensitivity. It is my practice to dilute the serum in 500 cc. of a solution of dextrose and sodium chloride and administer it intravenously by the drip method. The polyvalent serum is given daily for six doses.

For patients suspected of having allergic sensitivity, Vincent,[1] of Brussels, advocated the use of 7½ grains (0.49 Gm.) of sodium benzoate and 7½ grains (0.49 Gm.) of sodium salicylate at three hour intervals for two doses and then at an increased interval until one feels certain

1. Vincent, H.: Therapy by New Specific Serum, Bruxelles-méd. **16**:227 (Dec. 15) 1935.

that the patient will not have a severe reaction. When one is especially afraid that a severe reaction may occur, a large dose of sodium salicylate may be given intravenously.

Patients with septicemia due to Str. haemolyticus require an adequate amount of fluids, which can best be given intravenously in the form of a solution of dextrose and sodium chloride. Critically ill patients require from 3,000 to 4,000 cc. of this solution daily. It should be administered slowly by the continuous drip method. When a blood transfusion is required in the period of intravenous therapy, it can be given without removing the needle by placing a hemostat on the rubber tubing, temporarily stopping the flow of the saline solution. After the transfusion is completed, the hemostat is removed and the drip of the solution of dextrose and sodium chloride is resumed. This method of using the same venipuncture for the transfusion and for the administration of serum and the solution of dextrose saves the vein for future use.

The biologic measures which have been mentioned do not possess the efficiency shown by the various arsenical preparations in the destruction of Spirochaeta pallida. Since sulfanilamide and its derivatives were brought to the attention of physicians by Domagk, of Germany, and through the experimental and clinical work of Long and Bliss and others, the publicity given this drug through the lay press has caused the medical profession and the public to become much interested in its therapeutic value.

The clinical observations by many competent observers clearly indicate the potent chemotherapeutic action of this drug and its derivatives in the treatment of infection due to a hemolytic streptococcus and also to some extent in the treatment of other infections, namely, those due to a pneumococcus, the meningococcus or the gonococcus. The toxic effect of this drug is becoming more manifest with its continued use, and additional experience with these chemicals will be necessary before they can be properly evaluated as therapeutic agents.

COMMENT

Successful medical therapy depends on early and adequate intravenous administration of immunized blood, polyvalent antistreptococcus serum, solutions of dextrose and sodium chloride and sulfanilamide or its derivatives.

The assertion by some clinicians that blood transfusions are only supportive measures is no longer tenable in the light of more recent clinical observations, which clearly indicate that immunized transfusions

of whole blood promote phagocytosis, bacteriolysis, neutralization of soluble toxins and, lastly, bactericidal activity.

The transfusions of whole blood from a donor convalescent from surgical streptococcic infection is more desirable, at least theoretically, than therapy with scarlet fever serum. The duration of immunity in compatible donors convalescent from surgical streptococcic infection is not known. I have never observed a patient with a recurrent infection of the blood stream by a hemolytic streptococcus, nor have I reviewed reports in the literature. The surgeons of the hospitals of each city should make a permanent record of patients who have recovered from infection of the blood stream and secure permission for grouping, thus making them available as donors for emergency transfusions.

In addition to the biologic therapy mentioned, certain general measures, such as adequate feeding, rest, stimulants and proper elimination by the bowels and kidneys, must be carefully outlined from day to day.

CONCLUSIONS

1. Transfusion of immunized whole blood should be given for its prophylactic value to persons with lowered resistance, as indicated by the opsonic index and the blood picture.

2. In cases of infection of the blood stream and the fixed tissues, transfusion of immunized whole blood has a definite curative value and should be administered every other day until the well-being of the patient justifies discontinuance.

3. The treatment of infection of the blood stream secondary to infection of the middle ear and mastoid is primarily medical.

REPORT OF CASES

Notes on four patients treated surgically and with transfusions of immunized blood are as follows:

CASE 1.—G. V., a white youth aged 20, entered the hospital on March 1, 1936, complaining of pain in the left ear, sore throat and nasal discharge, which followed an attack of influenza of two weeks' duration. The tonsils were acutely inflamed. The frontal sinuses were clear. Both antrums were dark.

Three days after the patient's admission, acute otitis of the right ear developed, necessitating myringotomy. Despite active local and general treatment, the general condition progressed unfavorably, as shown by the laboratory and clinical data in table 1. On March 11 bilateral mastoidectomy was performed. The left mastoid was sclerotic but was definitely involved. The right mastoid was pneumatic and broken down. The patient continued to manifest evidence of sepsis, and one week after mastoidectomy a severe chill, elevation of temperature and a positive blood culture definitely pointed to infection in the blood stream. All the

TABLE 1.—*Data Concerning Case 1*

I. Laboratory Findings

A. Blood

Date	White Blood Cells	Red Blood Cells	Hemoglobin, Percentage	Differential Count, Percentages: Myelocytes	Metamyelocytes	Stab Cells	Polymorphonuclears	Eosinophils	Basophils	Lymphocytes	Monocytes
3/ 2/36	15,650	5,241,000	108	0.0	2.0	51.0	37.0	0	1	9.0	0.0
3/ 6/36	13,400	5,231,000	105	1.0	4.0	48.0	37.0	1	0	5.0	4.0
3/ 9/36	13,300	5,090,000	95	1.0	5.0	39.0	40.0	0	0	15.0	0.0
3/10/36	15,250	4,860,000	98	2.0	5.0	54.0	29.0	1	0	7.0	2.0
3/11/36	14,000	4,880,000	98	1.0	3.0	51.0	34.0	0	0	10.0	1.0
3/12/36	12,250	4,240,000	92	0.0	4.0	47.0	42.0	0	0	7.0	0.0
3/13/36	12,750	4,850,000	91	0.0	2.0	69.0	16.0	0	0	8.0	3.0
3/14/36	10,000	4,540,000	95	0.0	2.0	83.0	9.0	0	0	5.0	1.0
3/15/36	9,900	5,390,000	100	5.0	1.5	80.0	18.0	0	0	4.5	0.5
3/16/36	14,650	4,990,000	99	0.0	1.0	62.0	22.0	0	0	14.0	1.0
3/17/36	9,650	4,950,000	99	0.0	3.0	49.0	37.0	0	0	10.0	1.0
3/18/36	10,600	5,370,000	101	0.0	1.0	50.0	37.0	0	0	4.0	8.0
3/19/36	14,950	4,870,000	100	0.0	1.0	39.0	45.0	0	0	8.0	7.0
3/20/36	21,750	4,400,000	94	0.0	2.0	55.0	35.0	0	0	5.0	3.0
3/21/36	24,850	4,780,000	97	0.0	3.0	45.0	46.0	0	0	5.0	1.0
3/22/36	24,100	4,620,000	95	0.0	0.0	44.0	46.0	0	0	3.0	7.0
3/23/36	22,500	4,170,000	96	0.0	2.0	45.0	46.0	0	0	5.0	2.0
3/24/36	14,100	4,460,000	96	0.0	0.0	38.0	49.0	0	0	8.0	5.0
3/25/36	21,550	4,400,000	97	0.5	1.5	44.5	45.5	0	0	4.0	3.0
3/26/36	24,850	4,590,000	96	0.0	1.0	44.0	46.0	0	0	9.0	1.0
3/27/36	21,200	5,070,000	96	0.0	0.0	40.0	51.0	0	0	8.0	1.0
3/28/36	19,850	4,680,000	95	0.0	0.0	38.0	53.0	3	0	5.0	1.0
3/30/36	24,600	4,840,000	95	0.0	0.0	30.0	56.0	0	1	12.0	1.0

B. Urinalysis

Date	Color*	Reaction	Specific Gravity	Albumin	Sugar	Acetone	Diacetic Acid	Microscopic Examination
3/ 9/36	CY	Acid	1.014	1+	0	0	0	Occasional pus cell; few red blood cells
3/14/36	CY	Acid	1.008	1+	0	0	0	
3/15/36	CLA	Acid	1.025	1+	0	0	0	Occasional pus cell; many red blood cells; many hyaline casts
3/20/36	CA	Acid	1.020	1+	0	0	0	Occasional pus cell; few red blood cells; many hyaline casts
3/22/36	CA	Acid	1.025	1+	0	0	0	Occasional pus cell; no red blood cells
3/24/36	CA	Acid	1.020	0	0	0	0	No pus; no red blood cells; no casts

C. Bacteriology

3/ 2/36: Smear from left ear, many streptococci and pus cells
3/ 2/36: Smear from gums, negative for Vincent's bacillus
3/10/36: Smear from right ear, many streptococci and pus cells
3/12/36: Blood culture negative
3/18/36: Blood culture showed streptococci; culture from blood agar showed Str. haemolyticus
3/25/36: Smear from pus on hand showed many streptococci; culture from blood agar showed Str. haemolyticus
3/26/36: Blood culture showed no growth after four days' incubation; spinal fluid showed 2 white blood cells per cubic centimeter; pressure normal

II. Roentgen Findings

Right ear: Large pneumatic mastoid; cells broken down and cloudy throughout, with indications of mastoid suppuration
Left ear: Small, infantile, sclerotic, chronically diseased mastoid

III. Medical Treatment

3/ 7/36: 5% dextrose, 1,000 cc. intravenously
3/11/36: 5% dextrose, 3,000 cc. intravenously
3/12/36: Direct transfusion of whole blood, 180 cc.
3/14/36: 5% dextrose, 1,000 cc. intravenously; direct transfusion of whole blood, 210 cc.
3/16/36: Direct transfusion of whole blood, 125 cc.
3/18/35: 5% dextrose, 1,000 cc. intravenously; direct transfusion of whole blood, 150 cc.
3/19/36: Neoarsphenamine, 10 cc. intravenously
3/20/36: 5% dextrose, 1,000 cc. intravenously; direct transfusion of whole blood, 120 cc.
3/21/36: 5% dextrose, 1,000 cc. intravenously
3/23/36: 5% dextrose, 1,000 cc. intravenously; direct transfusion of whole blood, 120 cc.
3/26/36: Direct transfusion of whole blood from immunized donor, 120 cc.

IV. Surgical Treatment

3/ 1/36: Paracentesis of left ear
3/ 4/36: Paracentesis of right ear
3/11/36: Bilateral simple mastoidectomy
3/21/36: Both wounds reopened; sinus plates removed; lateral sinuses exposed and left sinus packed off; lumbar puncture
3/25/36: Abscess of right carpal region incised and drained
3/26/36: Abscess of left sacroiliac region incised and drained; abscess of upper part of left arm incised and drained
3/27/36: Two abscesses of left arm incised and drained
4/11/36: Abscess of right sacroiliac region incised and drained

* In all the tables CY means clear yellow; CL, cloudy amber; CA, clear amber; CLBY, cloudy bright yellow, and CLRY, cloudy reddish yellow.

recognized tests failed to fix definitely the portal of entry of the infection. The right sinus was freely exposed and found healthy; then the left was opened. It was covered with granulations, and a mural clot was found. The sinus was compressed. Improvement followed this procedure. During the following two weeks seven metastatic cutaneous abscesses developed, which were drained surgically. Seven blood transfusions were given, as shown in charts 1 and 2. The seventh was from a boy aged 10 years, possessing immunity to this infection

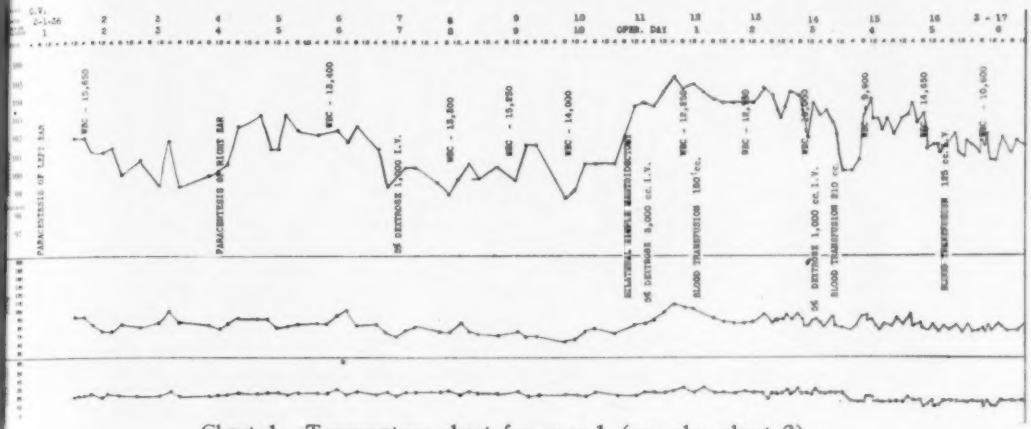

Chart 1.—Temperature chart for case 1 (see also chart 2).

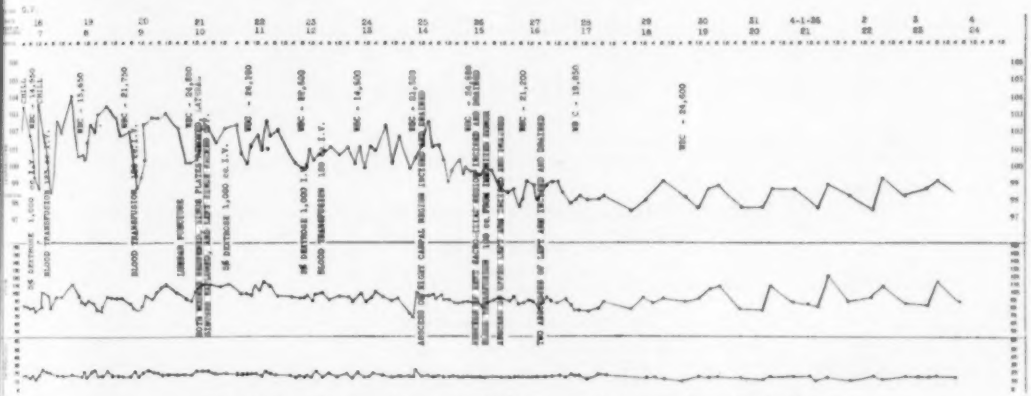

Chart 2.—Temperature chart for case 1 (see also chart 1).

because he had recovered from a similar infection three years before. All septic symptoms promptly subsided after this transfusion, and recovery was uneventful and permanent.

CASE 2.—L. R., aged 30, came to the hospital on March 13, 1936, complaining of pain in the left ear and mastoid area of two weeks' duration. The infection in the ear followed an attack of scarlet fever.

TABLE 2.—*Data Concerning Case 2*

I. Laboratory Findings

A. Blood

Date	White Blood Cells	Red Blood Cells	Hemoglobin, Percentage	Differential Count, Percentages						
				Myelocytes	Metamyelocytes	Stab Cells	Polymorphonuclears	Eosinophils	Basophils	Lymphocytes	Monocytes
3/14/36	14,250	4,750,000	93	0	4	29	39	1	0	25	1
3/21/36	20,500	4,410,000	82	0	2	42	44	0	0	11	1
3/22/36	19,200	4,100,000	85	0	3	45	42	0	0	7	4
3/23/36	13,350	3,870,000	84	0	1	47	34	0	0	15	3
3/24/36	10,100	4,240,000	86	3	0	65	20	0	1	5	0
3/25/36	12,700	4,900,000	90	0	2	50	33	0	0	13	2
3/26/36	8,550	4,550,000	87	0	2	36	43	0	1	12	6
3/27/36	9,800	4,370,000	85	0	2	28	50	0	0	15	4
3/28/36	10,150	4,270,000	86	0	1	35	36	1	0	18	9
3/30/36	13,050	4,300,000	84	0	3	24	43	1	1	13	5
3/31/36	22,100	4,420,000	89	0	3	40	36	0	1	15	5
4/ 1/36	15,650	4,160,000	83	0	0	34	42	1	1	15	7
4/ 2/36	17,750	3,950,000	82	0	1	39	49	0	0	9	2
4/ 4/36	10,050	3,860,000	81	0	2	44	25	0	0	24	5
4/ 6/36	10,350	3,820,000	81	0	2	33	32	0	0	28	5
4/ 7/36	16,000	4,710,000	90	0	1	35	40	2	0	19	3

B. Urinalysis

Date	Color	Reaction	Specific Gravity	Albumin	Sugar	Acetone	Diacetic Acid	Microscopic Examination
3/14/36	CLY	Acid	1.018	1+	0	0	0	Occasional pus cell; few red blood cells
3/14/36	CLY	Acid	1.019	1+	0	0	0	Occasional pus cell; few red blood cells
3/21/36	CLBY	Acid	1.026	1+	0	0	0	Occasional pus cell; many red blood cells
3/22/36	CLBY	Acid	1.030	3	0	0	0	Few pus cells; many red blood cells
3/23/36	CLRY	Acid	1.023	3	0	0	0	Few pus cells; many red blood cells
3/24/36	CLBY	Acid	1.027	2	0	0	0	Few pus cells; many red blood cells
3/25/36	CLY	Acid	1.012	2	0	0	0	Few pus cells; many red blood cells
3/26/36	CLY	Acid	1.010	1+	0	0	0	Occasional pus cell; few red blood cells
3/28/36	CLY	Acid	1.008	1+	0	0	0	Occasional pus cell; occasional red blood cell
4/ 1/36	CLY	Acid	1.016	1+	0	0	0	Occasional pus cell; occasional red blood cell
4/ 3/36	CLY	Acid	1.014	1+	0	0	0	Occasional pus cell; occasional red blood cell
4/ 7/36	CLY	Acid	1.012	1+	0	0	0	Occasional pus cell; occasional red blood cell

C. Bacteriology

3/21/36: Blood culture negative
3/23/36: Smear from lateral sinuses and from curet showed a streptococcus; culture made from syringe negative
3/24/36: Blood culture negative
4/ 2/36: Smear of pus from left arm showed a streptococcus; culture from blood agar showed Str. haemolyticus

II. Roentgen Findings

Right ear: Small infantile type of mastoid, apparently normal
Left ear: Large pneumatic type of mastoid, cloudy and diseased

III. Medical Treatment

3/15/36: 10% dextrose, 1,000 cc. intravenously
3/21/36: 5% dextrose, 1,000 cc. intravenously
3/22/36: 5% dextrose, 1,000 cc. intravenously; direct transfusion of whole blood, 210 cc.; 10% dextrose, 1,000 cc. intravenously; patient tested for sensitivity to streptococcus serum and 5 cc. of serum administered
3/23/36: Antistreptococcus serum, 15 cc.
3/24/36: Antistreptococcus serum, 10 cc.; 10% dextrose, 1,000 cc. intravenously; 3 units U 10 insulin
3/25/36: Antistreptococcus serum, 10 cc.
3/28/36: Antistreptococcus serum, 5 cc.
3/29/36: Antistreptococcus serum, 5 cc.
4/ 4/36: Direct transfusion of whole blood, 120 cc.
4/ 6/36: Lipoprotein preparation twice a day; direct transfusion of whole blood, 200 cc.
4/ 7/36: Lipoprotein preparation twice a day
4/ 8/36: Lipoprotein preparation twice a day
4/ 9/36: Lipoprotein preparation twice a day

IV. Surgical Treatment

3/13/36: Simple mastoidectomy
3/21/36: Wound reopened, lateral sinuses exposed and blocked off

Special examination revealed a profuse discharge from the left ear. The right ear was normal. Both maxillary antrums were dark. Physical examination gave negative results. The clinical, laboratory and roentgen examinations revealed positive evidence of suppuration of the left mastoid.

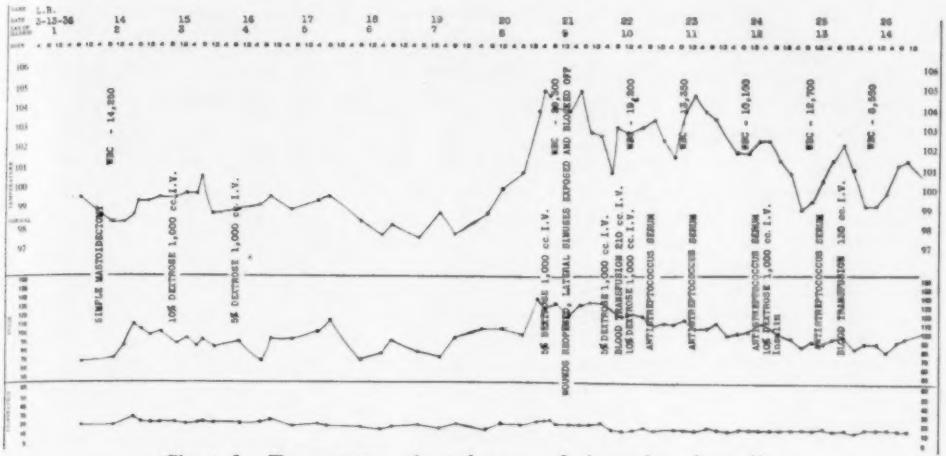

Chart 3.—Temperature chart for case 2 (see also chart 4).

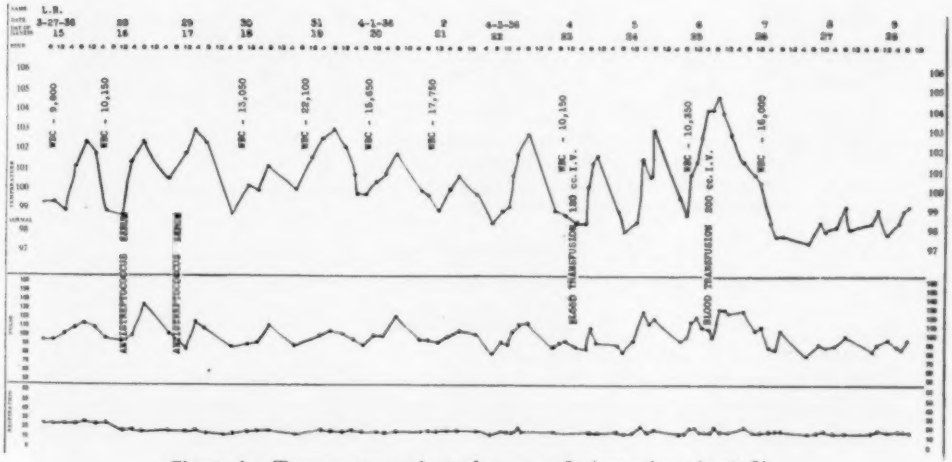

Chart 4.—Temperature chart for case 2 (see also chart 3).

On March 14 a simple mastoidectomy was done. The sinus plate had been destroyed by disease, thus exposing the sinus. The patient had persistent pain in the operative area, requiring opiates for relief. On the sixth postoperative day the temperature was 105 F., associated with an increased leukocyte count and a chill. On the seventh day the temperature again reached 105 F. At this time the

lateral sinus was exposed and compressed after free bleeding occurred from the bulbar portion. A culture made from blood from the sinus showed a hemolytic streptococcus. The patient continued to manifest evidence of sepsis. The temperature curve and the laboratory findings are shown in charts 3 and 4 and table 2. The medical treatment consisted of free utilization of antistreptococcus serum, dextrose and sodium chloride in solution and transfusion of blood from a compatible convalescent donor.

TABLE 3.—*Data Concerning Case 3*

I. Laboratory Findings

A. Blood

Date	White Blood Cells	Red Blood Cells	Hemoglobin, Percentage	Differential Count, Percentages							
				Myelocytes	Metamyelocytes	Stab Cells	Polymorphonuclears	Eosinophils	Basophils	Lymphocytes	Mononuclears
9/26/36	18,350	4,890,000	71	0	3	35	44	0	0	15	3
9/28/36	12,450	4,380,000	61	0	1	34	48	0	0	14	3
9/29/36	13,100	4,250,000	67	0	0	30	54	0	0	15	1
10/ 2/36	13,000	4,120,000	70	0	1	19	52	2	1	21	4
10/ 5/36	7,750	4,610,000	75	0	0	23	33	4	0	35	5
10/ 6/36	6,900	4,750,000	75	0	0	29	36	4	0	28	3
10/ 7/36	5,450	4,640,000	76	0	0	27	32	4	0	32	5
10/ 8/36	7,650	4,630,000	77	0	0	26	28	5	0	36	5
10/ 9/36	5,300	4,420,000	77	0	0	28	25	5	1	37	4

B. Urinalysis

Date	Color	Reaction	Specific Gravity	Albumin	Sugar	Acetone	Diacetic Acid	Microscopic Examination
9/26/36	CLA	Acid	1.026	1	0	0	0	Many pus cells; few red blood cells
9/28/36	CLY	Acid	1.027	1	0	0	0	Many pus cells; no red blood cells
10/ 6/36	CLY	Neutral	.....	0	0	0	0	No pus cells; no red blood cells

C. Bacteriology

9/26/36: Smear from left ear showed many pneumococci

9/27/36: Blood culture negative after three days

9/28/36: Culture from mastoid opening and canal of left ear showed mixed growth of pneumococci, streptococci and staphylococci

II. Roentgen Findings

Left mastoid cloudy, diseased, cells at periphery broken down

III. Medical Treatment

9/26/36: 5% dextrose, 1,000 cc. intravenously

9/27/36: Antistreptococcus serum, 12 cc. intramuscularly

9/28/36: Antistreptococcus serum, 12 cc. intramuscularly; 5% dextrose, 1,000 cc. intravenously; direct transfusion of whole blood from nonspecific immunized donor, 210 cc.

IV. Surgical Treatment

9/26/37: Simple mastoidectomy on left side

CASE 3.—C. H., aged 17, came to the hospital on Sept. 25, 1936, complaining of pain in the left ear and mastoid area of one week's duration. The infection in the ear developed as a sequel of tonsillitis.

Special examination showed that the right ear was normal. The canal of the left ear contained a thick purulent discharge indicating destruction of bone. Tenderness on pressure was elicited over the entire mastoid process. The blood cell counts, temperature, pulse record and laboratory findings are given in table 3 and chart 5. On the day following admission, the clinical symptoms of mastoiditis became more pronounced, with elevation of the temperature to 104 F. and definite roentgen findings. A complete mastoidectomy was done. On September 26 the

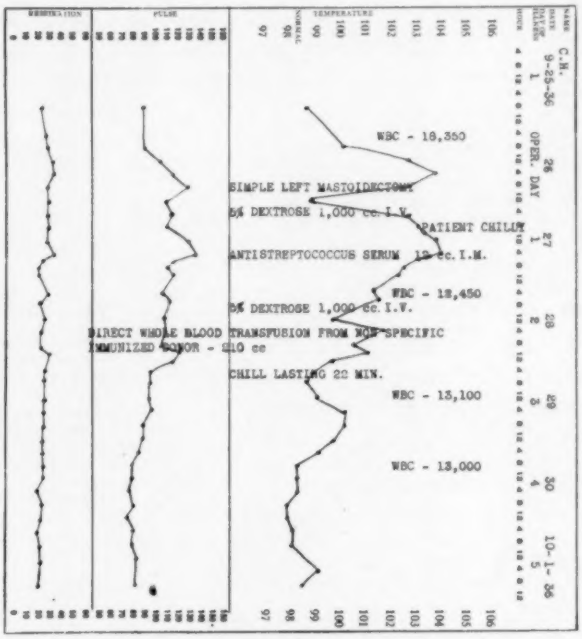

Chart 5.—Temperature chart for case 3.

entire mastoid process showed marked involvement, with suppuration. The sinus and dural plates were not disturbed, as they presented a normal appearance. The culture of material from the mastoid wound showed a mixed growth of pneumococci and streptococci. The temperature postoperatively was of a typical septic character, ranging from 99 to 104.2 F.

After a transfusion of nonspecific immunized blood, the septic symptoms promptly subsided and the temperature came to the normal level. This patient did not have a positive blood culture, but the septic symptoms definitely indicated a beginning invasion of the blood stream, all of which disappeared promptly after the transfusion of immunized blood.

CASE 4.—H. W., aged 15, came to the hospital on March 11, 1937, complaining of a persistent discharge from the right ear for about two and one-half weeks, which followed a spontaneous rupture of the tympanic membrane two days after the onset of pain in the ear.

TABLE 4.—*Data Concerning Case 4*

I. Laboratory Findings

A. Blood

Date	White Blood Cells	Red Blood Cells	Hemoglobin, Percentage	Differential Count, Percentages						
				Myelocytes	Metamyelocytes	Stab Cells	Polymorphonuclears	Eosinophils	Basophils	Lymphocytes	Monocytes
3/11/37	12,000	4,650,000	81	0	0	16	44	3	0	33	4
3/12/37	16,600	4,070,000	72	0	0	26	53	1	1	14	5
3/15/37	8,700	4,220,000	86	0	0	11	58	4	0	20	7
3/17/37	14,550	4,000,000	88	0	0	23	63	2	1	10	0
3/20/37	12,050	4,510,000	90	0	0	20	48	0	1	27	4
3/22/37	14,540	4,410,000	92	1	3	36	31	0	0	36	5
3/23/37	11,750	4,440,000	93	0	0	55	20	1	0	14	10
3/24/37	9,500	4,680,000	93	0	0	50	25	5	0	16	4
3/25/37	19,550	4,640,000	92	0	0	28	58	0	1	13	1
3/26/37	13,100	4,250,000	92	0	0	22	48	5	0	19	6
3/27/37	9,750	4,610,000	92	0	0	18	50	4	0	24	4
3/29/37	12,950	4,170,000	91	0	0	18	67	6	1	10	3

B. Urinalysis

Date	Color	Reaction	Specific Gravity	Albumin	Sugar	Acetone	Diacetic Acid	Microscopic Examination
3/11/37	CLY	Alkaline	1.020	0	0	0	0	Occasional pus cell; occasional hyaline cast
3/12/37	CLY	Alkaline	1.017	1	0	0	0	Occasional pus cell; no casts
3/17/37	CLY	Alkaline	1.015	1	0	0	0	No pus; no casts
3/23/37	CY	Alkaline	1.015	1	0	0	0	No pus; occasional hyaline cast
3/24/37	CY	Acid	.....	1	0	0	0	No pus; occasional hyaline cast
3/26/37	CY	Acid	1.020	0	0	0	0	No pus; no casts

C. Bacteriology

3/11/37: Smear from right mastoid, many streptococci
3/12/37: Culture from right mastoid on blood agar showed streptococcus

II. Roentgen Findings

Right ear: Densely cloudy, all cells diseased and broken down
Left ear: Normal

III. Medical Treatment

3/12/37 to 3/29/37: Sulfanilamide tablets every four hours
3/21/37 to 3/25/37: A total of 50 cc. prontosil intramuscularly
3/22/37: Direct transfusion of whole blood, 150 cc. from nonspecific immunized donor
3/24/37: Direct transfusion of whole blood, 150 cc. from immunized donor

IV. Surgical Treatment

3/11/37: Simple mastoidectomy performed on right ear; sigmoid sinus covered with granulations; sinus plate removed until healthy sinus was exposed

Special examination showed that the left ear was normal. The canal of the right ear contained a thick purulent discharge. Slight tenderness was present over the tip of the right mastoid. Roentgen examination showed definite evidence of suppuration of the bone of the mastoid area. General physical examination gave essentially negative results.

On March 11 a simple mastoidectomy was done. The dura and the sinus plate had been destroyed by disease, thus exposing the sinus, which was covered with granulations. The wound was left open, with the sinus protected by a piece of rubber dam. For six days postoperatively the patient's condition was satisfactory and the temperature was practically normal. On the seventh day, after a slight chill coincident with a sharp increase in the leukocyte count, the temperature rose from 98 to 103.8 F. On the following day the septic character returned and remained until the sixteenth day. An uneventful recovery followed. A transfusion of 150 cc. of nonspecific blood was given on the eleventh day. The clinical condition remained about the same. On the fourteenth day another transfusion of 150 cc. of whole blood was given, the donor being a former patient who had recovered from a similar infection about a year previously. After this second transfusion the temperature dropped to normal and remained so. Medical therapy included the use of sulfanilamide tablets by mouth and a derivative of sulfanilamide, known as prontosil soluble (the disodium salt of 4-sulfamidophenyl-2′-azo-

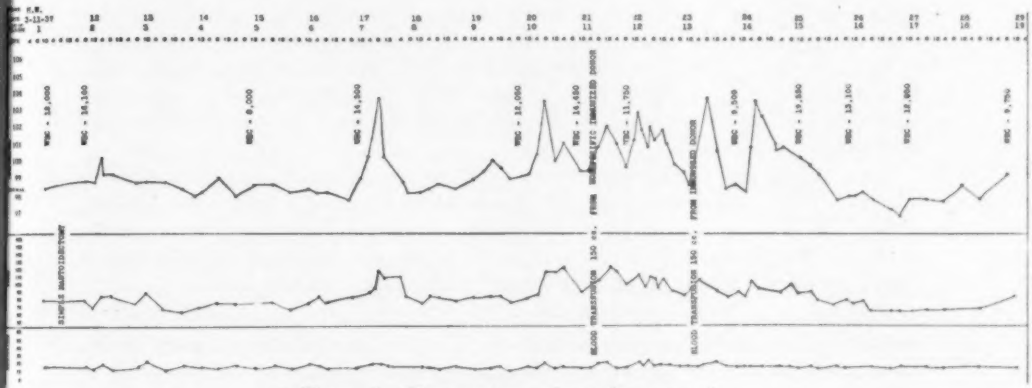

Chart 6.—Temperature chart for case 4.

7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonic acid), which was given intramuscularly.

ABSTRACT OF DISCUSSION

Dr. John A. Kolmer, Philadelphia: The cellular removal of micro-organisms in sepsis is largely dependent on the presence of certain antibodies in the blood, notably the opsonins and bactericidal agents, and is aided to some extent by the leukins and plakins from leukocytes and blood platelets. No wonder, therefore, that biologic therapy, into which blood transfusion properly falls, must continue to play an important part in both the prophylaxis and the treatment of septicemia. The most important principle in the treatment of surgical septicemia is to provide early and adequate drainage of the foci of fixed tissue infection. It is not difficult for organisms to be removed from the blood when one prevents their constant accession to this tissue from the primary foci. Therefore, early, prompt surgical drainage should stand foremost.

In addition to this primary and important surgical drainage, the physician must devote careful consideration to what he can do toward helping the hard-pressed patient in meeting this ordeal.

From the standpoint of both prophylaxis and treatment, I am thoroughly in favor of blood transfusion, not so much to furnish hemoglobin and erythrocytes as to furnish the things that are carried in the plasma, opsonins, bacteriolysins and complement as well as the nonspecific bactericidal substances called leukins and plakins.

I make a plea for the early administration of some of the newer antistreptococcus serums both from the standpoint of prophylaxis and from the standpoint of treatment. All the immune serums have far more prophylactic than curative value, and just as is true in the serum treatment of lobar pneumonia, diphtheria or mengococcic meningitis, so is it true with streptococcic septicemia that much depends on the early administration of serum in frequent and adequate doses.

Dr. Gill put much emphasis on the advisability of giving the patient the blood of a convalescent donor if one can be found, particularly in a case of infection due to a hemolytic streptococcus. If such a donor cannot be located, the next best choice is a person who has recovered from scarlet fever, because this too is a hemolytic streptococcic infection.

In addition, there is now available in sulfanilamide a chemical agent that renews one's hope from the standpoint of chemotherapy. This compound and its derivatives are not yet to be thoroughly relied on for disinfecting the foci of infection in fixed tissues. They have not yet fulfilled the ideal attained by arsphenamine in the treatment of syphilis, but they represent a hopeful advance, and their administration should be part of the therapeutic program in the treatment of streptococcic septicemia.

One should not forget the advantage of general measures—keeping up an adequate fluid intake, preferably by the administration of dextrose by bowel, giving adequate amounts of brandy or whisky, cardiac support, plenty of rest and if need be morphine, as it is much better to induce rest with this valuable agent than to allow the patient to exhaust himself with restlessness and pain.

DR. JAMES A. BABBITT, Philadelphia: Dr. Gill has presented his concern with a charmingly frank and courageous defense. He advocates a rather early operative intervention, and in the case reports cited there seems no doubt as to his operative judgment, but the title of his presentation must apply to hundreds of cases in which the infection is far less frank. May not transfusions of immunized blood during the preliminary period aid in determining the operative indication?

Most physicians will agree that in the aggregate more mastoidectomies have resulted badly from too early than from too late intervention.

Infection of the blood stream is a subtle matter. It has been stated that when cultures are made hourly bacteria will be found in the blood of a normal person at some time during the twenty-four hours, yet in that of a patient critically ill, with all the symptoms of bacteremia, organisms do not always appear. Surgical judgment must often depend on other criteria. Perhaps the presence of the organism is not the all-important guide.

There has been almost startling progress in the analysis of blood changes—analysis which seems to enable one to judge the status of the infection and to predict its probable course. The old conventional blood picture, even with the Schilling estimate, no longer stands. Maturation, degenerative changes in the immature cells, the percentage of toxic granules, changes in the cytoplasm and the nucleus and variation in the absolute count of lymphocytes, monocytes, eosinophils and basophils all enter the prognostic picture.

Dr. Gill is correct in denying the assured success of arsphenamine and quinine to the newer biochemical products, but in the splendid cooperative research studies

of today on the therapeutic evaluation of pool and lyophile serums, streptococcus serum from an immunized donor, nonspecific protein therapy, bacteriophage and even chemical adjuvants the products are gradually reaching the goal.

In the third case presented, antistreptococcus serum, dextrose and saline solution and convalescent blood serum were all used. The recovery of two of my patients was apparently due to sulfanilamide. In one case of secondary mastoiditis in which there was a cell count of 1,020 in the spinal fluid and symptoms of meningeal involvement reaching even the stage of respiration of the Cheyne-Stokes type, this was safely pushed to the cyanotic stage on several occasions. An early spinal injection of antimeningococcus serum and later blood transfusions may have been important factors, but the up and down variation in the progress of this critically ill patient seemed to be controlled by sulfanilamide.

In another patient who was far from a good operative risk, lyophile serum, immunized streptococcus serum and blood transfusion were adjuvant to routine biochemical aid.

This has been an interesting presentation, and the immunizing of donors may yet prove the most important procedure.

Dr. Earl Le Roy Wood, Newark, N. J.: Physicians should be most grateful to Dr. Gill for this direct paper containing so many practical suggestions and again drawing attention to the value of transfusions of blood containing antibodies and other immune substances specific against the infections they are attempting to control. There is little left to be said in discussion after Dr. Kolmer's and Dr. Babbitt's summary of the essential modern knowledge of this subject.

The case reports demonstrate the definite value of the blood transfused from persons who have recovered from similar infections. They reemphasize the importance of specific therapy, especially specific biologic therapy as a supplement to the surgical treatment. Universal specific therapy has long been the goal of all physicians, and every step toward it is a real advance. Dr. Gill's practical suggestion to utilize the antibodies of patients who have survived similar profound infections is novel. That they be made more readily available as donors of blood to new victims of the same infection is an excellent idea. This is direct specific biologic therapy. Emphasis should again be laid on the importance of determining the exact nature of every invading organism. This is a prime essential for specific therapy, whether biologic or chemical. With such information recorded, the person who has recovered is a possible donor of blood for another person suffering from the identical infection. The value of the immune substances in the new blood depends on its specificity. Attention should be paid not only to the type of organism but to the strain of the bacteria. In summation, this means that the nature of every infection should be determined exactly and recorded; the increasing use of specific therapeutic agents necessitates the reemphasis of this point.

At the Eye and Ear Infirmary in Newark, N. J., all the various remedies enumerated by Drs. Gill and Kolmer have been used with various degrees of success. Recently my associates and I have had our share of extraordinary results from sulfanilamide. However, blood transfusions have been our mainstay for the longest period, and we have been utilizing immunized donors for transfusions whenever possible. To this end we have placed our dependence on the freshly immunized amateur donor, utilizing for the stimulation of antibodies an autogenous vaccine. This method, while slower, is definitely specific. It requires some time, probably a minimum of two weeks, before there is any assurance of the presence of specific immune substances in the donor's blood. Considerable

time, however, can be saved by anticipating the emergency by preparing the vaccine and administering it in heavy doses to a prospective donor, preferably a relative, thus preparing him for an eventual blood transfusion.

Dr. Gill's suggestion that one be even better prepared for these emergencies by having available a list of donors with a detailed record of their previous infections is a valuable suggestion that I shall be more than ready to follow.

DR. E. G. GILL, Roanoke, Va.: I thank all three of the gentlemen for their discussion, and I appreciate the suggestion offered by Dr. Babbitt for further study along this line. That is what I hope to do, and I trust that something more will come from this mode of therapy.

FURTHER STUDIES IN THE HYDROGEN ION CONCENTRATIONS OF NASAL SECRETIONS

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The study of the hydrogen ion concentration of nasal secretions in various diseases of the respiratory tract was continued throughout the years 1934 and 1935. In all, 350 determinations were made on 125 subjects. The conditions presented were as follows: Six patients had atrophic rhinitis, 11 came for bronchoscopic treatment, 25 had colds and 83 had allergic rhinitis or allergic rhinitis with superimposed infection.

The hydrogen ion concentration was determined with the glass microelectrode. The description of the method and the precautions taken in its manipulation and in the handling of the secretion were given in an earlier paper.[1]

Tables 1 and 2 show the p_H for tracheobronchial secretions and atrophic rhinitis, respectively. The number of cases for each condition is too small to be used as a basis for definite conclusions, but the trend of the p_H is indicated. The secretions are primarily purulent and mucopurulent, and in consequence the hydrogen ion concentration is normal or on the acid side. The normal p_H of blood (7.4) is taken as the normal for the tissue. In general, the results substantiate the reports in the literature[2] that the p_H of a secretion decreases in value as the secretion changes from opalescent mucus to whitish mucopus to pale yellow through deep yellow and greenish yellow fetid pus. It is interesting to note in table 1 the occurrence of the pathogenic organism, Haemophilus influenzae, in 5 of the cases. The secretion from 3 persons showed no culturable bacteria; 2 of these did not have any bacteria in the stained smear. The microscopic examination of secretions from the third patient, G. J., showed many bacteria, but no growth was obtained. This lack of viability of the micro-organisms might be attributed to the high

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1. Buhrmester, C. C.: A Study of the Hydrogen-Ion Concentration, Nitrogen Content and Viscosity of Nasal Secretions, Ann. Otol., Rhin. & Laryng. **42**:1041 (Dec.) 1933.

2. Mittermaier, R.: Untersuchungen über die Wasserstoffronenkonzentration an Sekreten und Schleimhäuten, im besonderen bei chronischen Nebenhöhlenerkrankungen, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **127**:1, 1930.

TABLE 1.—*Tracheobronchial Secretions*

Patient	Diagnosis	*pH*	Bacteriology	Secretion	Microscopic Examinations			
					Eosinophils	Epithelial Cells	Neutrophils	Bacteria
B. F.	Bronchial asthma	7.05	No growth	Opalescent, few pale yellow flakes	++++	+++	+++	0
G. J.	Bronchial asthma	6.12	No growth	Yellow mucopus	+	+	++++	+++
T. F.	Pulmonary abscess	7.27	Haemophilus influenzae; Micrococcus catarrhalis; gram-positive diplococci	Fetid yellow pus	+++	0	++++	++
K. K.	Mediastinal abscess; chronic tracheal bronchitis	7.17	Streptococcus viridans; Haemophilus influenzae; large gram-positive diplococci	Mucopurulent	+++	+++	++++	+++
K. E.	Carcinoma of bronchus	7.36	.....	Pale yellow mucopus	±	++	++++	++++
P. C.	Allergic bronchiectasis, 10 weeks	7.50	No growth	Mucopus	++++	++++	+++	0
D. H.	Bronchiectasis, 12 years	6.60	.....	Yellow tenacious mucopus	+	0	++++	+
F. R.	Bronchiectasis, 4 years	7.51	Streptococcus viridans; Neisseria catarrhalis; Haemophilus influenzae	Whitish yellow mucopus	+	+++	++++	+
V. J.	Bronchiectasis, 7 years	7.37	Haemophilus influenzae	White tenacious mucopus	±	+	++++	+++
P. A.	Bronchiectasis	7.45	Haemophilus influenzae; few gram-positive diplococci	Mucoid	..	..	..	..
P. W.	Bronchial asthma	7.60	Friedländer's bacillus, type A	Opalescent	+	++++	+	+

hydrogen ion concentration (p_H, 6.12). In an earlier paper I [1] reported that bacteria could not be cultured from an exudate when the p_H was below 6.4.

Colds have been divided into three groups in respect to the course and the nature of the secretion. In the first group are those that last only three or four days. In these the nasal discharge is usually available on the first day only. The secretion is thin and watery, contains much salt and little mucin and has a p_H of 7.97 to 8.11. To illustrate the second group, data from 2 typical cases are given in table 3 (patients H. A. & H. B.). In these cases the character of the secretion changes from a profuse, thin, watery fluid to scant, thick, clear mucus. The hydrogen ion concentration in the initial stages is within the alkaline range, dropping toward a normal value as clinical improvement takes place. This is in agreement with the findings of Hilding [3] that the p_H

TABLE 2.—*Atrophic Rhinitis*

			Microscopic Examination			
Patient	p_H	Secretion	Eosinophils	Epithelial Cells	Polymorphonuclears	Bacteria
M. G.	5.62	Greenish yellow mucopus	..	..	..	..
M. M.	6.36	Greenish yellow mucopus	+	+	++++	++++
	7.38	Thin watery mucus				
G. C.	6.15	Deep yellow mucopus	+	+	++++	++++
D. H.	7.13	Pale yellow mucopus	0	++	++++	++++
M. H.	7.11	Pale yellow mucopus	0	++	+++	+++
	6.20	Fetid greenish pus	..	++	++++	++++
V. T.	7.41	Pale yellow mucopus	+	+++	++++	++++

became elevated in the first four days of a cold and then fell slightly below normal in the purulent stage. The third group is made up of long protracted colds complicated by secondary infection, and the data were obtained in the outpatient clinic. At the time the patient was first seen, the cold was usually a week old, and a definite secondary infection had occurred. The case of patient A. T. is typical. He had a cold accompanied with bilateral maxillary sinusitis. In this condition the p_H may fall below the chemical neutral point, the discharge is usually mucopurulent and in some instances frank pus is observed.

The hydrogen ion concentration of the nasal secretion may reflect the constitutional status of the person. It has been stated that the p_H can be predicted from the gross appearance of the secretion. However, a constitutional factor may so modify the p_H as to make the statement ambiguous. This study presents the fact clearly. R. I., who was subject to hay fever, entered the outpatient clinic in September. The reaction

3. Hilding, A.: The Common Cold, Arch. Otolaryng. **12**:133 (Aug.) 1930.

TABLE 3.—*Colds*

Day	*pa*	Secretion	Condition	Bacteriology	Microscopic Examinations: Polymorphonuclears	Mononuclears	Lymphocytes	Epithelial Cells	Eosinophils	Bacteria
Patient H. B.										
1	8.08	Thin, clear, watery	Mucosa slightly red; not swollen	.....	±	0	0	++++	0	0
3	8.15	Clear mucus	Right side better; left red but not much swollen	.....	+++	0	+++	++++	0	0
5	7.50	Opalescent	Pharynx dark red; left side slightly swollen	.....	++++	0	0	++++	0	0
7	7.00	Deeply opalescent	Mucosa slightly red	Hemolytic and nonhemolytic Staphylococcus albus	++++	0	0	++++	++	0
9	7.43	Clear mucus	Mucosa almost normal; pharynx moderately injected	.....	++++	+	+	+++	++++	0
11	....	None	Patient discharged	Hemolytic streptococcus and staphylococcus	..	..	..	..	..	..
Patient H. A.										
2	7.93	Clear with few white flakes	Mucosa red, slightly thickened	.....	++++	+++	+++	+++	±	+
3	7.58	Clear with more white flakes	Mucosa red; not greatly swollen; pharynx diffusely injected	Friedländer's diphtheroids	++++	++++	+++	±	±	0
4	R 7.82 L 7.97	Slightly opalescent Clear	Mucosa on right side slightly red; mucosa on left side very red, no swelling; moderately injected pharynx	.....	++++	+++	+	+++	±	0
5	7.63	Clear and thicker	Pharynx still injected	.....	+	0	0	+	0	0
7	7.43	Clear mucus	Mucosa almost normal	.....	+++	++	+	++	0	0
9	....	None	Mucosa normal	.....	+++	0	0	++++	+	0
Patient A. T.										
7	7.10	Pale yellow mucopus	Mucosa red; not edematous	Hemolytic streptococcus; Staphylococcus albus	++++	+	±	0	+	++
10	R 6.87	Pale yellow mucopus	....	Hemolytic streptococcus	++++	+++	±	..	+	+++
	L 7.12	Pale yellow mucopus	....	.....	++++	++	++	+	+	+++
12	R 7.68	Clear	.....	.....	++++	+	+	+	+	++
	L 7.50	Opalescent								
14	R 7.80	Clear mucus	.....	.....	++++	+++	+	..	+	+
	L 7.96	Clear mucus	.....	.....	++++	±	..	0	±	++
17	R 7.06	Yellow mucopus	.....	.....	++++	++++	++	..	++++	+
	L 7.34	Pale yellow mucus	.....	.....	++++	+++	++	0	++	0
20	R 7.48	Pale yellow mucopus	.....	.....	++++	++	++	0	++	+
	L 7.68	Very pale mucopus								

of his secretion before treatment was alkaline. Melons, onions, garlic, nuts and all condiments were removed from his diet on September 21, as these were thought to aggravate his condition. When the skin tests were completed on October 12, cabbage, all fried foods, coffee, beans and peas were removed from the diet. The patient complained after the first elimination of food that it was hard to get enough to satisfy his appetite, as all the members of his family were fond of heavily spiced foods. The week after the removal of the more essential foods the patient reported the loss of 10 pounds (4.53 Kg.). The p_H of the secretion at this point was 6.51, although the character of the discharge was grossly identical with that of the previous secretion at a p_H of 7.76.

In starvation acidosis ketosis develops. It is reasonable to assume that nasal secretions would be one way to throw off acid radicals to

TABLE 4.—*Allergic Rhinitis Accompanied with Loss in Weight*

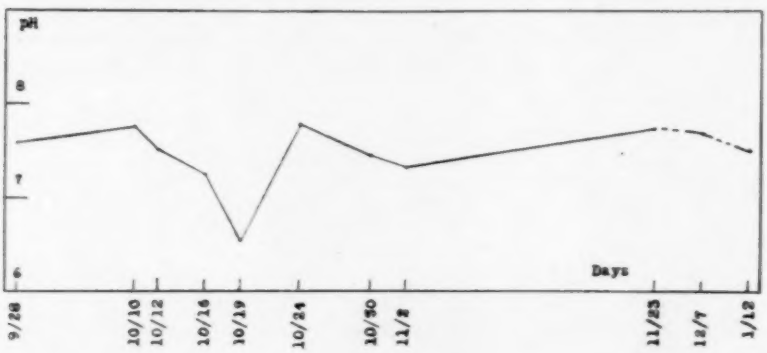
Patient R. I. Date	p_H	Secretion
9/28	7.60	Opalescent
10/10	7.76	Clear with some opalescent material
10/12	7.51	Clear with opalescent clumps; tenacious
10/16	7.24	Opalescent, thick and tenacious
10/19*	6.51	Clear; less tenacious than secretion on October 16
10/24	7.62	Clear; slightly opalescent
10/30	7.43	Clear; slightly opalescent
11/ 2	7.31	Clear; slightly opalescent
11/23	7.72	Clear
12/ 7	7.70	Clear; few flakes
1/12	7.50	Clear; opalescent

* The fall in p_H to 6.51 on October 19 is attributed to loss in weight.

maintain neutrality. The p_H of the blood was not taken, but the literature shows that probably no discrepancy would have been detectable at this point. As soon as the readjustment to the new food habits was made, the p_H returned to its original level. Table 4 and the chart give the data concerning this case in detail. A similar unusual hydrogen ion concentration was observed during the course of a common cold in a medical student. The p_H was: first day, 7.86; second day, 6.86; fourth day, 7.08, and fifth day, 6.53. The appearance of the secretions did not warrant such findings. On questioning, it was found that the patient was losing weight and had within the past five months lost 33 pounds (14.94 Kg.) on a supervised reducing diet. A third but less striking case was observed in the clinic. This patient had a food allergy. The p_H of the nasal secretion was 6.9 and 7.3 and was lower than that usually found in a similar condition. It was discovered that preceding the first determination the patient had lost 5.5 pounds (2.5 Kg.); before the second, 2 more pounds (0.9 Kg.) were lost in the two week period. Fourteen days later, when the patient had gained 1 pound (0.45 Kg.),

the p_H was 7.6. These 3 instances further substantiate the conclusion of Mittermaier [2] that the nasal secretion is affected both by a local and by a constitutional factor.

Whenever it was possible, the secretion was obtained from each naris separately. Many times, as seen in table 5, considerable variation occurred in the p_H. These differences were greater in colds than in allergic rhinitis with superimposed infection and least in allergic rhinitis. It was not uncommon to have a patient complain that one side of his nose ran profusely for a day and the other side did not begin to drip until the third or fourth day. In colds the two sides of the nose seldom showed an identical picture simultaneously. Dr. F. H. Borg, an otolaryngologist, followed the clinical picture of all the colds in which



Hydrogen ion concentration in allergic rhinitis accompanied with a loss in weight.

the secretions were tested. A pooled sample, particularly in a cold, reflects only roughly the state of irritation in the nose.

Table 6 shows data on typical cases of uncomplicated allergic rhinitis. The secretions were alkaline or normal, depending on the quality and quantity of the discharge. Table 7 presents data on cases of allergic rhinitis with superimposed infections of varying severity. The increase in infection is roughly paralleled by an increase in the hydrogen ion concentration. F. L. had a mild infection; R. H. a prolonged chronic infection, and R. L. a deep-seated more acute infection. The hydrogen ion concentration in each case can be correlated closely to the gross appearance of the exudate. It is only when secretions are affected by an unusual constitutional state, illustrated in table 4, that they are so modified that the p_H will not grossly correlate with the appearance of the discharge.

It should be recognized that the nasal secretion in amounts sufficient to collect is the result of an abnormal activity of the mucosa and that

the hydrogen ion concentration of the discharge is a result and not a cause of stimulation. However, as soon as the highly alkaline material is released, there is an increased permeability due to the change in the colloidal structure of the cell and a change in the electrostatic charge

TABLE 5.—*Various Conditions* *

Colds			Allergic Rhinitis			Allergic Rhinitis with a Superimposed Infection		
p_H Right Naris	p_H Left Naris	Difference	p_H Right Naris	p_H Left Naris	Difference	p_H Right Naris	p_H Left Naris	Difference
7.4	7.9	0.5	8.1	7.6	0.5	6.4	6.9	0.5
8.0	7.1	0.9	7.6	8.1	0.5	7.5	7.8	0.3
6.9	7.1	0.2	7.3	7.6	0.3	8.2	7.8	0.4
7.2	7.7	0.5	7.4	7.7	0.3	6.5	7.1	0.6
7.1	6.7	0.4	7.9	8.2	0.3	7.5	7.8	0.3
7.9	7.1	0.8	8.0	7.7	0.3	7.6	7.1	0.5
8.3	7.4	0.9	7.6	7.9	0.3	7.5	8.2	0.7

* The hydrogen ion concentration shows different conditions existing in the two nares when the secretions are collected simultaneously. Each pair of determinations represents one subject.

TABLE 6.—*Allergic Rhinitis*

Patient R. W.		Patient I. K.			Patient R. H.	
			p_H			
Date	p_H	Date	Right	Left	Date	p_H
	7.65					
1/29	7.45	1/25	8.25	8.18	1/28	7.56
2/ 3	8.10	2/ 3	8.07	7.56	2/ 3	8.10
2/ 7	7.98	2/ 8	7.82	8.19	4/ 6	7.67
2/11	7.40	2/25	....	7.95	6/24	7.78
		3/ 5	7.83	....		
		6/23	....	8.18		

TABLE 7.—*Allergic Rhinitis with Superimposed Infection*

Patient F. L.		Patient R. H.			Patient R. L.		
			p_H			p_H	
Date	p_H	Date	Right	Left	Date	Right	Left
3/ 4	6.40	2/ 6	6.54	6.32	2/ 7	7.84	8.18
3/ 5	8.18	2/ 8	6.80	5.86	2/13	6.78	6.80
3/ 6	6.94	2/11	....	6.42	2/20	7.45	7.79
3/ 7	7.12	2/13	7.06	6.22	2/23	7.90	7.05
3/ 9	7.08	2/20	6.91		6/23	7.73	
3/13	7.12	2/26	6.55				
4/ 1	6.50	2/27	7.45	7.13			
		3/ 5	6.38				

on the tissue. The ever present pathogenic organisms may then gain entrance into the mucosa and call forth neutrophilic infiltration. The discharge is now composed of a secretion, an exudate and a transudate. One cannot doubt the deleterious action of the thin watery secretion when the excoriation about a profusely running nose is remembered.

This study indicates that the initial physiologic response of the nasal mucosa is the same regardless of the etiologic stimulus, be it virus or allergen. In each instance a thin, watery, highly alkaline secretion is obtained. The clinical picture of the mucosa is dissimilar during this stage. In epidemic colds the mucosa is red and only slightly swollen, while the allergic nose shows a pale edematous mucous membrane. What course the disease takes will depend on the type and viability of the organisms present and on the natural or acquired resistance of the patient to these organisms. A good nutritional state of the body certainly increases the resistance to invading organisms.

SUMMARY

A study of 125 cases of disease of the upper respiratory tract was made.

Loss of weight is reflected in the nasal secretion by an increase in the hydrogen ion concentration.

The secretion from a cold is alkaline in the initial stage, and the p_H decreases to the normal as clinical improvement takes place.

The initial physiologic response of the nasal mucosa to stimulation is similar regardless of the inciting agent.

A CASE OF GANGRENOUS OSTEOMYELITIS OF THE PARANASAL SINUSES

ALFRED LEWY, M.D.
CHICAGO

REPORT OF CASE

B. J. P., a girl aged 11 years, was first seen by me on July 10, 1935. There was a swelling in the left cheek, extending into the lower eyelid and obliterating the upper part of the nasolabial fold. The girl's color was strikingly white. The history given was that following scarlet fever in March 1935 there was a discharge from the left side of the nose, associated with a swelling in the left cheek. After about a month of unsuccessful local and general treatment, a naso-antral window operation was performed. The discharge and swelling continued, and a peculiar musty odor of the breath developed, with fever. Physical examination revealed nothing of consequence except a daily temperature of 101 F. The Wassermann reaction of the blood was negative. A biopsy made at the time of the operation was reported as showing granulation tissue only.

When I first saw the patient, there was present, in addition to the swelling described, an ulcerated area about 5 by 5 mm. in the left side of the hard palate, covered with a whitish gray exudate and not involving the bone macroscopically. The mucosa bordering this area was elevated into several small ridges; the left upper canine tooth was loose, and the left nostril was filled with greenish mucopus. The mucosa of the right nostril was congested. Transillumination showed the left antrum dark. Roentgen examination revealed a similar condition, plus some clouding of the left ethmoid region. Smear of the material from the ulcer showed a predominance of fusiform bacilli and spirilla. Culture showed that there was a mixed infection. Another negative Wassermann reaction of the blood was obtained at this time. The patient was admitted to the Illinois Eye and Ear Infirmary on July 11.

The preoperative diagnosis was osteomyelitis of the maxillary bone, possibly a malignant condition. A Caldwell-Luc operation on July 15 revealed a thickened and somewhat softened facial wall, which was widely removed; the antral mucosa was moderately thickened and edematous and was dissected out in toto, although no free pus was found in the mucosa or bone. The original antral window was found to be adequate. Specimens of the bone and mucosa were sent to the National Pathological Laboratory and to Dr. I. Davidsohn at Mount Sinai Hospital, as well as to our own laboratory, for histologic diagnosis. All the pathologists agreed essentially that the tissue was inflammatory, with perhaps some resemblance to that involved by syphilis. Dr. Davidsohn's report was as follows:

"Sections show a mucous membrane the glands and the surface lining of which show no abnormalities. The stroma is hyperemic and infiltrated with many neutrophilic leukocytes, some eosinophilic leukocytes, lymphocytes, and a strikingly large number of plasma cells. In some areas the infiltration with leukocytes is pronounced; in others the leukocytes are mixed with fibrin and fill out areas where the native tissue is necrotic. The infiltration with plasma cells

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is particularly marked in certain areas. Some of them are multinucleated. Small multinucleated giant cells are present occasionally. Some granulation tissue, as well as some newly formed connective tissue, is seen. The picture is that of a chronic recurrent inflammation. The diagnosis is chronic inflammation (recurrent)."

Repeated Wassermann and Kahn tests of the blood of the patient and of both parents were negative.

Smears and cultures of material from the affected parts and of the secretions were examined by Dr. Virginia Fisher, at the University of Illinois, especially for fungi. No pathogenic fungi were observed.

Bacteriologic investigations were made by Dr. R. D. Herrold and Miss Wolsey, whose report was as follows:

"Direct smear from washings showed streptococci, fusiform bacilli, a few clusters of a blastomycete and other bacilli and cocci. Cultures, aerobic and anaerobic, showed a pure growth of hemolyzing streptococci and of blastomycetes.

"Inoculations of rabbits and guinea pigs gave the following results: Guinea pig A was given intracutaneous and subcutaneous injections of washings on December 6; on December 12 there was a necrosed area 12 by 15 mm. and 5 mm. deep. A nodule at the site of the subcutaneous injection was aspirated and cultured. Hemolyzing streptococci and blastomycetes were recovered in pure culture, and fusiform bacilli and spirilla were present in smears of this pus. Use of the Ziehl-Neelsen stain on original material and smears from the necrosed area gave negative results. Guinea pig B reacted similarly. Both animals recovered and were entirely healed by December 25. Guinea pig C was inoculated with washings previously treated with 3 per cent hydrochloric acid to eliminate extraneous bacteria. A hardened mass appeared at the site of intracutaneous injection, which healed spontaneously. Guinea pig D was inoculated with the pure culture of hemolyzing streptococcus previously mentioned. In forty-eight hours the region was inflamed and elevated, but it healed without necrosing. Material aspirated from this area showed a hemolyzing streptococcus. Guinea pig E was inoculated with a pure culture of the blastomycete mentioned. Elevation and inflammation without necrosis resulted. Aspirated material from the nodule showed the pure blastomycete. The nodule disappeared, and the guinea pig recovered. The rabbit was killed after treatment, and autopsy was performed; the result was the same as for guinea pig A. Nothing conclusive was found."

Because of the suggestion by Dr. Richard Jaffé, pathologist at Cook County Hospital, who saw the child during life, a special effort was made to culture the anaerobic necrophilic hemolytic streptococcus, but without success.

The local treatment, because of the predominance of the Vincent-Plaut organisms in the smears and the inconclusive results of cultures, was use of sodium perborate paste for the lesion in the mouth and irrigations of the antrum with solutions of the same drug. There was gradual improvement of the ulcer of the hard palate, but the necrotizing process continued in the other affected parts. The patient was given a high vitamin diet plus halibut liver oil and viosterol. Arsenicals were given without result; then antimony and potassium tartrate, and bismuth tartrate. Potassium iodide was also given. Four roentgen treatments seemed to make the patient's condition worse. The irrigations were changed to trinitrophenol and calcium carbonate, this being supposed to represent the chemical equivalent of the maggot treatment.[1] This treatment was later supplanted by use of copper sulfate. A 0.1 per cent solution of potassium chlorate was also tried. On July 23 the first blood transfusion, 300 cc., was given. On August 10 and a

1. Stewart, J. P.: Surg., Gynec. & Obst. **58**:155, 1934.

few days thereafter there was epistaxis. There appeared to be a little improvement in the patient's condition, in that her temperature dropped to a daily high of about 100 F. and the necrotic process seemed to have come to a relative standstill. She was permitted to go to her home in a suburb, where the antisyphilitic treatment was renewed.

On Oct. 8, 1935, the patient was readmitted to the Illinois Eye and Ear Infirmary. She had become weaker and more anemic during her stay at home. A bony slough had been removed from her nose on two occasions, and her septum, unaffected before, had been attacked by the necrotizing process. Her condition grew progressively worse, with remissions following blood transfusions, and her temperature began to fluctuate from normal to as high as 104 F. On November 3 a small lesion appeared in the left nasolabial region, covered with a honey-colored crust, which on exfoliation left a black excavation communicating with the antrum and progressively enlarging until the entire facio-antral wall, the naso-antral wall, the left alar portion of the nose and part of the septum were removed by sloughing, as seen in the photograph. Because of better nursing and

A, appearance of patient in November 1935; *B*, appearance of patient in December 1935.

consultation facilities, the patient was transferred on December 7 to the Children's Department, Cook County Hospital, where I continued in charge. Before the transfer she was seen by Dr. Joseph Beck, to whom the lesion appeared clinically to be syphilitic. Later at the County Hospital she was seen by Dr. Richard Jaffé, the pathologist, who suggested that the lesion was probably due to a necrotizing type of streptococcus.

On December 7 the heart was found to be enlarged and displaced to the left, and the resonance of the left side of the thorax was impaired. Roentgen examination showed a mottled infiltration of the upper half of the left lung. Two pediatricians believed that pneumonia had developed. (These observations were explained when autopsy revealed hydropericardium and atelectasis of the basal parts of the upper and lower lobes of the left lung, and bilateral hydrothorax.)

When I took charge of the case, there was a leukocyte count of about 12,000, but when the patient was admitted to Cook County Hospital in December the white cell count had dropped to an average of 4,000 and the red cell count to 3,000,000. Except for fluctuations following blood transfusions, the number of all cells had diminished steadily, in proportion, as had the hemoglobin content. On December 12 there was bleeding, requiring packing, which recurred several times; finally, sudden uncontrollable bleeding on December 17 resulted fatally.

The conditions observed at autopsy were as follows: gangrenous osteomyelitis of the left maxillary sinus with exposure of the sinus; a similar condition of the left ethmoid and sphenoid sinuses, suppurative otitis media and mastoiditis on the left side, catarrhal otitis media on the right side; severe general anemia; bloody gastric content; infective hyperplasia and softening of the spleen; hydropericardium and bilateral hydrothorax; compression atelectasis of the basal parts of both lobes of the left lung.

COMMENT

Otitis media had not been overlooked during the patient's lifetime but was considered incidental. The mastoiditis consisted merely of pus in the mastoid cells. An interesting side phenomenon was that this child while in bed grew to nearly 6 feet (180 cm.) in length, possibly owing to pituitary irritation. She merely grew tall and slim.

Under the title "Progressive Lethal Granuloma," Stewart [2] reviewed a number of cases, ten of which appeared similar to this and in all but one of which the patient died—in one four months after apparent cure of generalized sarcomatosis cutis and in one as a result of phthisis four years afterward. The only case on his list that occurred in this country was one of Wood's,[3] that of a woman with mutilating granuloma of the nose and face, with fatal ending. Hill [4] reported a case in which he diagnosed as sporotrichosis a condition which caused a necrotizing process. Joisten [5] reported under the name "granuloma gangrenosa" two cases in which the condition corresponded in nature and in course to that in the case described here. Joisten quoted Berendes [6] as having gathered twenty-eight cases from the literature but erred in stating that the patients were all males. Wood's patient and mine were females. Wood said that the condition resembles noma, except that no medical or surgical treatment avails. Joisten also tried sulfanilamide, his cases being more recent than mine. His pathologic report practically corresponded to mine, and he also failed to isolate a causative organism, although he made a careful effort. He stated that he suspected a symbiosis between the Vincent-Plaut organism and another or other organisms.

Differential diagnosis takes one far afield: to malignant processes, syphilis, tuberculosis, agranulocytosis, mycosis, myiasis, yaws or frambesia, leprosy, rhinoscleroma, rhinopharyngitis mutilans, trophic postencephalitic ulceration, anthrax and glanders. Various names have been given to what appears to be a process similar to that described. Le Chatelier, quoted by Stewart, called it lupus pernio.

25 East Washington Street.

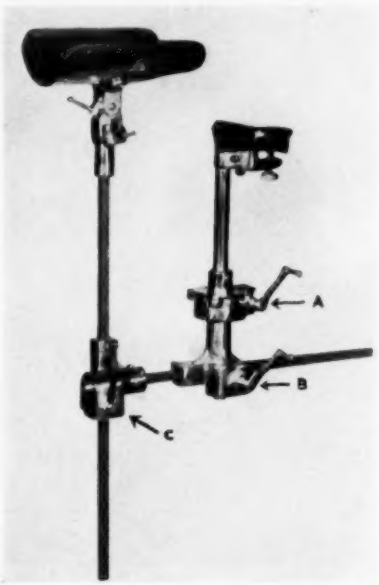
2. Stewart, J. P.: J. Laryng. & Otol. **48**:657, 1933.
3. Wood, George B.: Tr. Am. Laryng. A. **53**:63, 1931.
4. Hill, F. T.: Tr. Am. Laryng. A. **52**:148, 1930.
5. Joisten, E.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:105, 1936.
6. Berendes, cited by Joisten.[5]

Clinical Notes

A NEW TYPE OF HEAD REST FOR ENDOSCOPIC EXAMINATION

BERNARD WELT, M.D., BROOKLYN

There has been no improvement over the method developed by Boyce for steadying the head as a preliminary to the introduction of a rigid tube into the trachea or the esophagus. The advantages gained in the use of this position in



Photograph of the device. *A* controls the right and left motion; *B*, the forward and backward motion, and *C*, the upward and downward motion.

endoscopic examinations need not be entered into here. They have been amply discussed by Jackson and his workers.

For those who do not have the services of an assistant trained in holding the head according to the Boyce method, I wish to submit a description of a head holder which achieves the results obtained by that method as nearly as is mechanically possible. I have used this device since 1933 and have found it a useful aid in endoscopic examination with adults and with children over a certain age. With infants and young children, one needs an assistant to hold the head. This instrument is also useful in the performance of cerebellar decompression preliminary to the evacuation of a cerebellar abscess.

The device, which is illustrated in the accompanying photograph, consists of two vertical rods. One of these, the anterior rod, is attached to the table by

means of a clamp and has at its midportion a worm-gear that controls the right and left motion of the posterior vertical bar, which supports the head rest proper. At the lower extremity of the anterior vertical bar there is another worm-gear, through which runs a horizontal bar or rack, which varies the distance of the head rest from the table. Thus, the head rest can be adjusted to fit different patients. At the posterior extremity of the horizontal bar there is a third worm-gear, which controls the distance of the head rest below or above the table. The head rest proper is adjustable forward and backward and laterally, to the right or to the left. The head rest proper is shaped so that the nose, the mouth and the eyes are accessible to inspection during the administration of an anesthetic when the patient is in the prone position, as for cerebellar decompression.

This device is easily attached to any standard operating table. The position of the head can be assured; it can be changed at will to meet the varying needs of the operator, with a smoothness that only a trained assistant can give and without the need of continuous locking of the instrument in the position desired.

Since 1933 the head rest has been in use at the Jewish Hospital and at the Greenpoint Hospital in Brooklyn.

135 New York Avenue.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

FUNCTIONAL EXAMINATION OF HEARING

ROBERT SONNENSCHEIN, M.D.
AND
NORMAN LESHIN, M.D.
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During the past year we have seen more papers on the functional testing of hearing in the German literature than have been apparent for some time. As we noted several years ago, articles in German had become scarce, and the literature in the United States and England had by far overcome the lead which the Germans had many years ago held in so predominant a fashion.

Kelley [1] presents the historical aspects of bone conduction. Conduction of sound through the cranial bones was known as early as the sixteenth century. Ingrassia was one of the first to report on it, as was discovered in his work, edited in 1603, after his death. Capivacci in 1589 applied the knowledge of bone conduction to clinical otology by having the patient hold one end of a rod between his teeth, with a vibrating string of a musical instrument in contact with the other. If the patient heard the tone he had disease of the drum membrane, and if he could not he had disease of the labyrinth. Schelhammer in 1684 proved experimentally that the sound conveyed through the teeth is transmitted by the cranial bones and not by way of the eustachian tube.

Thomas Willis (1622-1675) established that the organ of hearing is in the cochlea. In 1704 Valsalva reported that the labyrinth was filled with liquid and air, but Cotugno in 1760 proved that it is filled only with liquid. In 1769 Scarpa discovered the membranous labyrinth. Jorissen in 1757 made a practical application of hearing by bone conduction in the use of the Jorissen stem, probably the first hearing aid of the bone conduction type. Wheatstone observed conditions in which one's sensitivity for bone-conducted sound can be increased, as by closing the external auditory meatus. He also demonstrated the lateralization phenomenon, which was confirmed by Weber in 1834.

1. Kelley, N. H.: Historical Aspects of Bone Conduction, Laryngoscope **47**:102 (Feb.) 1937.

Rinne in 1855 described the procedure for comparing air and bone conduction. Conta in 1864 showed the practical value of the tuning fork in testing hearing.

Schwabach described his observations in 1884 and 1885. Kelley also mentions the more recent contributions and observations pertaining to bone conduction contributed by Runge in 1923, Downey and Fowler in 1925 and Hallpike in 1930. The work of Pohlman and Kranz, Knudsen and Jones, Dean and Fowler brings the historical report up to the present.

The Committee of the American Otological Society on Methods of Testing the Hearing by Bone Conduction [2] emphasizes that bone conduction tests made without proper precautions yield incorrect observations and that the hearing problems presented by patients cannot be solved by the use of unreliable methods of examination. The two sources of sound which can be satisfactorily used in these tests are tuning forks and the bone conduction receivers of the electric audiometers. Forks of the frequencies of 435 and 512 d. v. are preferred for the best results. However, the committee suggests that bone conduction tests be made also over as wide a range of frequencies as possible, to obtain all additional data which might be of value in the future, with due regard to the possible sources of error. They suggest further that tests be made above the minimal threshold, at levels of intensity at which they are heard by normal persons. This furnishes a practical basis for predicting the ability of a patient to understand speech or any noises with a hearing aid. The committee also emphasizes the importance of a quiet, and even sound-proof, environment for testing. When room noise is present, proper allowances can be made; the committee refers to reports by British observers on methods for determining the masking effect of this noise. It stresses the importance of masking and urges that this measure be used as a routine when acuity of hearing is examined by means of tuning forks or audiometers. Several methods for masking are described, the simplest being blowing a stream of air across the external auditory meatus. Good steel tuning forks should be used, and the technic of alternate placements of the fork is recommended. The committee suggests that the total cycle of each alternation should not exceed five seconds for the lower frequencies and three seconds for the higher. A standard blow should be employed for activation, one which causes the prongs to vibrate at the maximum amplitude without damaging the fork. A simple method is to employ a metal rod covered with rubber tubing, to prevent dam-

2. Report of the Committee of the American Otological Society on Methods of Testing the Hearing by Bone Conduction, Ann. Otol., Rhin. & Laryng. **45:** 800 (Sept.) 1936.

age to the fork. This is pivoted at one end and permitted to swing from the horizontal to the vertical position before striking the tuning fork held in its path. The stem of the fork should be placed on the part of the mastoid which produces the loudest tone for the patient. This should be determined in a preliminary test.

The committee suggests that each otologist determine for his own forks the normal times for air and bone conduction by averaging data for a group of young adults with normal hearing. The rate of damping, expressed in decibels, should be determined for each fork, so that the degree of impairment in any given patient for that fork, expressed in decibels, can be determined. The method by which this can be done is described more fully in the report. The committee suggests that the determination be made with the use of different technics of handling for each fork. This has the advantage that the rate of decrement of each fork is determined by the Bureau of Standards, where a special apparatus gives a reading which is correct only for the method by which the fork is handled at the Bureau.

The committee mentions certain advantages and drawbacks of audiometers for bone conduction work. Measurements of thresholds can be made more quickly with the audiometer because a sound can be maintained at a given intensity and results can be checked at given intensities. Thresholds for a greater range of frequencies can be tested with an audiometer than with tuning forks in the short period allotted to this part of the examination. Furthermore, most audiometers and some bone conduction receivers make available a greater range of both frequencies and intensities than do tuning forks. The objections to audiometers are in general: their expense; the impossibility of quick repair; the possibility of inaccuracy without awareness of the examiner, and the fact that more accidents can happen to audiometers than to tuning forks. The specific drawbacks of audiometers for bone conduction tests are: First, there is lack of assurance that any of the bone conductor receivers now on the market will hold calibration for a number of years, and, second, different receivers of the same make generate markedly different amounts of acoustic energy for the same flow of current. Therefore, every otologist using a bone conductor receiver must check its calibration at frequent intervals. Finally, enough data are not now available to interpret observations made with bone conduction receivers as well as otologists think they can interpret the results of good tests made with tuning forks. The committee believes that manufacturers will succeed in overcoming these difficulties. When they do, all otologists will use audiometers for both air and bone conduction tests. Then there will be a real uniformity of method and a common language for bone conduction tests.

McNally and his associates [3] made a comprehensive study of bone conduction in cases in which a definite intracranial lesion was proved. In 37 of the 50 cases the presence of an intracranial lesion was confirmed. They came to the following conclusions: In 22 of the cases there was some loss of hearing which was in no way typical of the lesion in the brain, except in the 4 cases of tumor of the eighth nerve. In these cases there was complete nerve deafness on the side of the lesion, which was satisfactorily demonstrated only by masking the normal ear. In 4 of the 6 cases in which a cerebellar tumor was removed there was some loss of the high tones not typical of the cerebellar lesion. In 7 of the 9 cases in which a cerebral tumor was removed there was also a loss of hearing which could not be associated with the lesion. In cases in which there was loss of high tones the lesion could not be distinguished from early cochlear degeneration. The audiograms in these cases were of three types, showing an abrupt loss of high tones in the region of 4096 d. v., a gradual loss of high tones or a slip in the audiogram at 4096 d. v. Encephalography and ventriculography had no effect on the hearing in cases in which tests were made before and after the procedures.

Bone conduction was tested with the bone conduction attachment of the 2A Western Electric audiometer, with a tuning fork and with the monochord. No one method proved more reliable than another. The Hartmann tuning fork of 512 d. v. was found to be most suitable, and a uniform method of activating the fork proved a great advantage. When one uses a 2A audiometer, the monochord is necessary for checking the upper tone limit of hearing. Tests made with the tuning fork, the monochord and the audiometer must be accompanied by masking. The buzzer type is suitable for use with the tuning fork and audiometer, but a current of air may be necessary to mask the monochord. The absolute bone conduction test is a necessary part of any test for hearing. From their investigation, McNally and his associates found that bone conduction tests made with notes in the vicinity of 512 d. v. give the most reliable results. In some cases increased bone conduction was found when obvious nerve deafness was present or when the eighth nerve on the side of increased bone conduction was known to be severed. No explanation for this finding could be given. The Weber test, which was carried out in most cases, proved to be misleading. The investigation showed not so much that any one test is better than another but that examination must be complete and thorough. To estimate bone conduction accurately, careful tests should be made with the audiometer, tuning fork and monochord. These should

3. McNally, W. J.; Erickson, T. C.; Scott-Moncreiff, R., and Reeves, D. L.: Clinical Observations on Bone Conduction, J. Laryng. & Otol. **52**:375 (June) 1937.

be accompanied by air conduction tests on all three instruments. Suitable masking is necessary; careful history taking, physical examination and vestibular tests are often indispensable. Hearing tests in this series of cases proved of no value in localizing the intracranial lesion, except in cases of tumor of the eighth nerve.

Macfarlan,[4] in discussing bone conduction, brings out the fact that in making a bone conduction test not only is there direct transmission of sound to the end-organ but skull resonance is set up, which may greatly alter the findings. This resonance is influenced by the asymmetry of the skull, the density of the bone and the condition of the sinuses. In addition, bone conduction tests are subjective, depending on the observations and responses of the patient. Macfarlan suggests the use of skull phonendoscopy as an adjunct to the present methods of testing. A method of performing this test is the use of binaural auscultation tubes placed in the patient's and the observer's ears. With the use of this method and with the vibrating fork on the patient's vertex, the following observations were made:

Normal patients reported that they heard sounds as long as the examiner, and equally in the two ears. Asymmetry of the skull or unilateral empyema of the frontal or maxillary sinuses threw the resonance to the affected side. Impacted cerumen or occlusion of the canal lateralized the sound for the patient to the side of the lesion, but the examiner heard less well from the affected side; in unilateral mastoid disease usually, but not always, the sound for both the patient and the examiner was referred to the affected side. Bilateral disease of the sinuses or ears gave no significant findings. In uncomplicated unilateral nerve deafness the sound was referred to the better ear for the patient, but was equal in the two ears for the examiner. Uncomplicated unilateral catarrhal deafness lateralized the sound for both the patient and the examiner to the diseased ear.

Subjective determinations of bone conduction on normal persons with use of a fork of 512 d. v. showed that when the handle of the fork was placed lightly in the external auditory meatus, resulting in both bone and air conduction, the sound was perceived longest. The next longest duration was obtained from the upper teeth, the next from the antrum of the mastoid and the shortest from the vertex. To ascertain objective findings, only skull resonance was checked on the dried skull. With an activated resonator screwed in the occiput, auscultation was made at various points on the skull. Resonance was weakest at the vertex and was markedly increased as radial lines were followed to the dense parts of the skull. This was first thought to be due to the heavi-

4. Macfarlan, Douglas: Skull Resonance, Ann. Otol., Rhin. & Laryng. **45:** 768 (Sept.) 1936.

ness and density of the bones. However, masses of plaster or plasticine attached to the inside of the vault also increased the resonance at these points. The inference can be drawn that a tumor attached to the bone would do likewise. One should keep in mind the vagaries and contradictions of skull resonance and skull auscultation which make these tests unreliable for absolute findings. However, findings on auscultation are frequently of value as confirmatory evidence.

One of us (R. S.) [5] reviewed the work of Schoen and Goldberger, who also employed a double phonendoscope connecting the examiner's ears with the external auditory canals or the skull of the patient, in order to test head resonance. They used this method also to check subjective statements in certain tuning fork tests.

Löwy [6] states that the ability of the patient to hear with bone conduction aid cannot be prognosticated by tuning fork tests. These are usually made at a pitch of 128 d. v., while 1000 d. v. is necessary for many formants of speech. Löwy advises the use of his electric apparatus for testing at 1000 d. v. The sound of tuning forks at this pitch is carried over to the other ear.

Zangemeister [7] states that the type of deafness depends on whether the endolymph is under positive or negative pressure. In the hypertonic labyrinth there is loss for high and low tones and for whisper and conversation. The condition is difficult to distinguish from otosclerosis with loss of bone conduction. The labyrinthine reactions are normal. In the hypotonic form there are little loss of hearing and slight loss for the c-4 fork and the caloric test, but bone conduction is approximately normal. Treatment for the hypertonic form is administration of 40 per cent magnesium sulfate, hypertonic solution of sodium chloride or a 50 per cent solution of dextrose daily, for eight or ten days. In the hypotonic form there are often vertigo, tinnitus and low blood pressure; then, neosynephrin or ephedrine is advised.

Reger,[8] in testing both normal and impaired ears at levels slightly above threshold, found that the hard-of-hearing ear perceived a louder sensation of sound than the normal ear, with the same frequency and the same number of decibels above its threshold. This is even more

5. Sonnenschein, Robert: Functional Examination of Hearing, Arch. Otolaryng. **3**:60 (Jan.) 1926.

6. Löwy, K.: Beobachtungen mit Knochenleitungshörapparaten, Monatschr. f. Ohrenh. **71**:531 (May) 1937.

7. Zangemeister, H. E.: Zur Differentialdiagnose und Therapie der Innenohrschwerhörigkeit, München. med. Wchnschr. **84**:253 (Feb. 12) 1937.

8. Reger, Scott N.: Difference in Loudness Response of the Normal and Hard-of-Hearing Ear at Intensity Levels Slightly Above the Threshold, Ann. Otol., Rhin. & Laryng. **45**:1029 (Dec.) 1936.

evident when the loss of hearing is more pronounced. The magnitude of the auditory sensation apparently increases more rapidly at levels of intensity just above threshold.

Fowler [9] presents a preliminary report on experiments to determine a method for the detection of threatened or potential ankylosis of the stapes before there is any loss of hearing as shown by the usual functional tests. This is a study of sounds well above minimum audibility. The ear does not listen to sounds of faint intensity. In ordinary conversation the normal ear is accustomed to sounds from 40 to 60 decibels above normal minimal audibility. That some persons appear not to hear loud sounds, and therefore do not hear with hearing aids as well as one would expect from their threshold audiograms suggested the possibility that there may be lesions which interfere proportionately less with faint sounds than with louder sounds. On the other hand, other persons may hear better than their audiograms imply, and loud sounds may disturb some markedly deafened persons as much as those with good hearing. This implies the possibility that lesions interfere more with faint sounds than with louder sounds. That otosclerosis often appears suddenly suggests a critical incident in the growth of the lesion, such as definite involvement of the annular ligament.

To investigate these phenomena, both the minimal audible intensity and the above-threshold intensity for normal and subnormal ears must be investigated. Hearing by both air and bone conduction should be measured. The patient's ears must act as reference, and the reference tone has been proposed as 1,000 cycles at 70 decibels above the normal threshold. The object of the test is to determine whether an ear hears sounds at various intensities above threshold better or worse than would be expected from threshold audiograms and whether there is a boosting or a restraining effect at certain intensities in certain ears.

Sounds of known loudness are thrown alternately into the ears, the patient indicating in which ear the sound is louder or whether it is of equal loudness in the two ears. If there is a difference, the loudness is varied in the worse ear until it equals that in the better ear. This balance is called the alternate binaural loudness balance. When there is a difference, it is measurable and signifies the additional number of decibels required to produce the same sensation of loudness in the worse ear as that in the better ear.

The experiments revealed four distinct phenomena: First, certain persons show a constant difference at all intensities; second, certain persons show a changing difference at different intensities; third, a combination of the first and the second phenomenon may exist, and

9. Fowler, Edmund Prince: A Method for the Early Detection of Otosclerosis, Arch. Otolaryng. **24**:731 (Dec.) 1936.

fourth, anomalies in discrimination of intensity occur; i. e., in certain ears at critical intensities small changes appear greater or less than they do to the average normal ear or to the opposite ear. Fowler shows a graph to illustrate each of these groups. The method of graphing is not wholly satisfactory. The author hopes this method of testing may have considerable practical application and may point a way to better understanding of auditory function.

Ciocco [10] presents data on acuity of hearing by bone conduction in 516 patients (896 ears) whose hearing by bone conduction was better than that by air conduction. This analytic study was made to determine whether acuity of hearing by bone conduction bears a relationship to such factors as age, the appearance of the tympanic membrane, tinnitus, a history of familial deafness and the degree of impairment of hearing by air conduction. Bone conduction time was prolonged in 26.7 per cent of the cases, shortened in 7.2 per cent and normal in 66.1 per cent. The tests were made with a tuning fork of 512 d. v. Hearing by air conduction in persons with prolonged bone conduction time was similar on the average to that in persons with normal bone conduction time, but was more acute than that in patients with shortened bone conduction time. A normal tympanic membrane was observed with greater frequency in the ears having prolonged bone conduction time, while a perforated tympanic membrane was found more often in those with shortened bone conduction time. The frequency of tinnitus was practically the same for both groups. A history of familial deafness was considerably more frequent in cases of prolonged than in those of shortened bone conduction time. This finding indicates an association between prolonged bone conduction time and familial deafness. The effect of age on bone conduction appears to be the most noteworthy and significant finding. Persons with prolonged bone conduction time were on the average the youngest, and those with shortened bone conduction time, the oldest. "Prolonged bone conduction time in all probability represents the upper 'tail' of the frequency distribution of the normal variation." The fact that prolonged bone conduction time was found more frequently in the younger persons confirms the hypothesis that acuity of hearing by bone conduction is associated with some factor or factors other than the condition of the cochlear nerve and end-organ.

Shambaugh Jr.[11] raises the question of efficiency of the audiometer for diagnosis and whether the old tuning fork tests are needed or can be entirely supplanted. He describes a series of cases showing the difficulty of accurate diagnosis from audiometric determinations alone.

10. Ciocco, Antonio: Conduction Deafness, Arch. Otolaryng. **24**:723 (Dec.) 1936.

11. Shambaugh, George E., Jr.: The Interpretation of Bone Conduction and Other Tests of Hearing, Ann. Otol., Rhin. & Laryng. **45**:779 (Sept.) 1936.

The shortcomings of the audiometer in tests for conduction deafness are as follows: The audiogram in cases of conduction deafness gives a false picture of hearing for low tones because it introduces an element of bone conduction into hearing by air conduction. Therefore, it cannot be used with the bone conduction curve for diagnosis. This is because the standard receivers fit snugly against the ears. A pure air conduction curve can be obtained only by holding the standard receiver close to, but not touching, the ear. When this is done an air space is introduced, which results in marked diminution in the loudness of the sound. It is also difficult to hold the ear phone at exactly the same distance from the ear throughout the test. Also, the increased diffusion of sound into the outer air passes to the better-hearing ear, giving a false picture of the hearing in the poor ear when there is a great difference between the two ears. The last objection can be overcome by proper masking.

Shambaugh shows the various types of conduction deafness giving rise to different kinds of hearing tests. When the movements of the stapes alone are impeded, there is marked loss for the low tones, very little loss for high tones and an early negative reaction to the Rinne test. When the movements of both the stapes and the round window membrane are involved, either by occlusion of the external canal or by fluid in the middle ear, there is more equal loss for both low and high tones, while in the Rinne test a shortened, positive or equal, conduction time, instead of a negative result, may be obtained. The diagnosis of these various types can easily be made by means of the tuning fork tests. Shambaugh shows the difficulty of differentiating nerve deafness for low tones with diplacusis from conduction deafness, which is important and can be done only by use of the tuning fork tests. Diplacusis is easily demonstrated by the tuning forks, and the Schwabach test is of paramount importance for diagnosis. Nerve deafness for low tones with diplacusis is due to exudative labyrinthitis and can usually be relieved by removing a focus of infection. Since the drum membranes are normal and the eustachian tubes are patent, a diagnosis of otosclerosis might easily be made if it were not for the tuning fork tests.

He concludes that the audiometer cannot be used for diagnosis to the exclusion of the tuning fork tests. Considerable time is required for thorough audiometric determinations, and the necessity in some cases for extreme accuracy does not permit delegation of this work to a technician. The length of time required for these tests introduces the element of fatigue. Opportunities for error are apt to appear more frequently with a complicated mechanical device than with the simple, direct tuning fork tests. Shambaugh further suggests that in performing the Schwabach and Rinne tests one should at frequent intervals remove

the fork from the skull, or in the Rinne test pass it from the skull to the meatus, to avoid auditory fatigue and auditory after-images. He also suggests that since in case of a conduction lesion the inner ear is already in a sound-proof environment, owing to the exclusion of outside sounds by the lesion, the Schwabach test should not be performed in a sound-proof room. In fact, the noisier the environment, the more positive the results in the Schwabach test in cases of conduction deafness. The article is accompanied by many audiograms explaining and proving the points brought out in the paper.

Fowler and Fowler [12] report findings on bone conduction as measured with a 2A Western Electric audiometer on 614 hospitalized patients with good hearing. The subjects who showed a loss or a difference in the two ears of 15 or more sensation units for air conduction were regarded as not having normal hearing and were not included in the test. The audiograms were charted by taking an average for each decade of life and using the ear with the better hearing for air conduction. The typical rise was found at 2048 d. v., as was also demonstrated by Bunch and Ciocco. For all decades of life remarkably good hearing was shown at 256 d. v. There was a drop at 1024 d. v., but the average hearing was good for all decades. At 4096 d. v. there was a sharp drop for both air and bone conduction, but less for bone than for air conduction. The drop at 8192 d. v. was not as well marked as that at 4096 d. v., except in the latest decades. The graphs would show a greater loss at the highest frequency if the 2A audiometer were more powerful.

Patients with good hearing in the late decades show that age per se is not the cause of deafness. There was little variation of hearing between the sexes, women having slightly better hearing than men. There were small variations between the disease groups. The group of rheumatic patients were the worst. This may be due either to the history of past infection of the respiratory tract or to the salicylate therapy. The good bone conduction as compared with the poor air conduction for low notes in the later decades suggests that the most important type of deafness for all ages is the conduction type. There was usually nerve impairment for the higher frequencies in the old patients, but the conduction loss was the greater. This suggests that the etiology of deafness in old persons, as well as in children, is infection of the upper respiratory tract. With more opportunity for infections of the middle ear in older persons, more and denser scar tissue is formed at strategic points. This scar tissue is the cause of impaired hearing, and not damage to the cochlea, as is generally supposed.

12. Fowler, Edmund P., and Fowler, Edmund P., Jr.: Normal Hearing by Bone Conduction as Measured with an Audiometer, Ann. Otol., Rhin. & Laryng. **45**:754 (Sept.) 1936.

Kafka [13] presents the electro-acustotestometer for bone conduction tests and describes the advantages of this instrument. The tuning fork is activated through an electromagnet operated by the patient by use of a push-button. The stop watch simultaneously records the length of time the sound is heard by bone conduction and is stopped by the patient pressing another button when he no longer hears it. With the use of this method, uniform stimulation of the fork is obtained; the factor of interference with the use of the examiner's hands is eliminated, and the error of comparison with the examiner's hearing is removed. A mean average for the time of vibration of each fork has previously been determined.

One objection which can be offered with reference to the employment of this apparatus is that there is probably considerable distraction to the patient entailed in the use of the push-buttons.

Witting [14] gives information intended to acquaint prospective purchasers with some of the salient points in audiometers. The purity of the tone is important. If no analysis is given with the audiometer, the presence of overtones near threshold intensity should be tested by listening. Instruments operating on alternating current may have a hum which vitiates measurements at the higher frequencies. The audiometer is reasonably safe if the hum threshold is at least 50 decibels below the normal thresholds for all frequencies above 8192 d. v. for persons with normal hearing at the low tones. Unique hearing loss scales should be avoided. The sensation unit or decibel scale should be used. The response of the receiver on a given audiometer should remain constant to an amount less than the intensity step of the intensity control. If the bone conduction receiver is to be adequate, the air-radiated sound should be of lower intensity than the bone conduction perception as measured on a subject with good air conduction hearing.

The operation of an audiometer is important. Witting suggests that the test be started by using frequencies of the middle range, and then carrying first upward from that point and then downward. This is done since the patient experiences less trouble with the middle frequencies. In testing frequencies below 256 cycles, care should be taken that the patient holds the receiver close to the ear; otherwise slight leaks may occur, giving lower readings. When one feels that holding the receiver close to the ear introduces an element of bone conduction, a cap may be used over the receiver. Caps of sponge rubber with a semi-smooth finish seem to give the best results. One should know whether

13. Kafka, M. Martyn: Electro-Acusto Testometer: Its Value for Accurate Timing and Recording of Bone Conduction Tests, Arch. Phys. Therapy **18**:507 (Aug.) 1937.

14. Witting, E. G.: The Selection and Operation of Audiometers, Laryngoscope **46**:747 (Oct.) 1936.

the audiometer has been calibrated for the cap on the receiver when this is included with the instrument. If that has not been done, the examiner can determine the difference produced by the cap by testing several normal subjects with and without it. When testing above 4,000 cycles, one should look for the phenomenon of standing waves, which is neutralization of the original wave by the reflected wave of the head. This phenomenon can be eliminated by moving the receiver slowly toward and away from the ear of the patient. When the maximum intensity is reached, the patient signals. If removing the receiver from close contact with the ear causes the examiner to suspect that the sound is heard in the opposite ear, that ear should be masked. Masking is important. Witting also suggests the use of a sound-proof chamber as a valuable adjunct to the otologist's office. An absolutely sound-proof room is difficult to provide in professional offices in the buildings in business centers of large cities. However, by special treatment of the walls with celotax, felt, etc., or by using specially constructed doors and double windows, fairly satisfactory rooms may be had, without excessive cost.

Firestone [15] presents his method of passive masking, which he finds efficient. The procedure is simply to have the patient in one room and the audiometer in an adjoining room. The intervening wall prevents the patient's receiving sound waves by air conduction directly from the instrument. By means of the push-button signal, the patient informs the examiner when he hears the sound. This eliminates the need of cumbersome, and rather questionable, masking apparatus. Firestone has found that this procedure has yielded more dependable audiometric findings than before.

Ersner [16] presents the relative merits of audiometric and tuning fork tests. He demonstrates that in normal persons air conduction is not always twice that of bone conduction. Of a series of 200 normal persons, Ersner and his co-workers found a normal ratio in 55 per cent, diminished bone conduction (Schwabach test) in 39 per cent and a marked loss in bone conduction in 6 per cent. Tuning forks are of little value in testing persons with a hearing loss of 50 per cent or over, especially when frequencies of from 128 to 2048 d. v. are employed. The frequencies above 2000 d. v. are of little value, because they are not readily perceived by bone conduction. In testing high frequencies the audiometer is more accurate, as the tone can be sustained and the pitch intensified. The average tuning fork tests will generally give sufficient data for a diagnosis of the type of deafness. Ersner calls attention to the necessity for calibrating the attenuator with the audiometer and the

15. Firestone, Charles: Passive Masking as an Aid in the Use of an Audiometer, Laryngoscope **46**:924 (Dec.) 1936.

16. Ersner, Mathew S.: Relative Merits of Audiometric and Tuning Fork Tests, Arch. Phys. Therapy **18**:503 (Aug.) 1937.

receiver with which it is to be used, to make it mathematically and scientifically accurate. Most audiometers have not taken into consideration the standardization of the receiver. Therefore, as sound is transmitted through the receiver there is an error in the calibration, due to the nonuniform frequency response of the receiver. The receiver should likewise be standardized with the audiometer. Ersner also mentions the new type of audiometer introduced by Knudsen and Jones, which determines the type of hearing aid best suited to the patient.

Wells [17] brings out the fact that insufficient attention has been paid by the state to the so-called deaf and that justice will not have been done until they have been considered and provided for to an extent at least equal to that for the blind. Regular testing of hearing by the gramophone audiometer has been established in the ordinary schools of London for several years, and many thousands of children have been tested. If one applies the figures for deafness to the entire population of England and Wales, one estimates that about 6,500,000 persons, or 16 per cent of the population, have defective hearing in one or both ears, or approximately 1 in every 6 persons. The number of persons who would be covered by a Deaf Pension Act, now proposed and under consideration, would be in the neighborhood of 150,000. This group includes persons with a hearing loss of about 60 decibels with the pure tone audiometer. The method employed in this investigation was the application of the incidence found in "samples" of various sections of the population to the whole population of these sections.

Shurly [18] makes a plea for the prevention of deafness and urges proper propaganda and more funds to carry out a necessary program. In Detroit a card system has been instituted in the office of each principal, for each school child. On the card the name of the family physician is noted. In this way more direct, and quicker, contact with the family physician and parents and better follow-up are possible. Classes for the saving of hearing should be started. Knowledge of the heredity of deafness and the danger of intermarriage of deaf mutes and of consanguinity should be disseminated and proper legislation taken. Approximately 61 per cent of deafness is the result of suppurative disease of the ear. All efforts should be utilized to build up resistance against infections causing this condition. Proper treatment for suppuration of the ear and conditions of the sinus and throat is important. The problem of immunity should be studied, and hygiene, with application to proper clothing in and out of school, and proper diet should be considered. Shurly urges that otologists joint hands with the League for the Hard

17. Wells, Arthur G.: An Estimate of the Incidence of Defective Hearing in England and Wales, Brit. M. J. **2**:18 (July 3) 1937.

18. Shurly, Burt R.: Prevention of Deafness in the School Child, Ann. Otol., Rhin. & Laryng. **46**:223 (March) 1937.

of Hearing to educate the people and the profession in the best known scientific facts concerning the prevention of deafness.

Newhart [19] stresses that from the otologist's point of view the hearing problem of first importance in education is the prevention of hearing deficiencies and the conservation of hearing among school children. The resolution was passed in 1926 by the House of Delegates of the American Medical Association to encourage the general use of scientifically accurate methods for the routine testing of hearing of all school children, namely, by means of the audiometer. The city and state of New York have been leaders in accepting these principles and incorporating these methods in their respective school health programs. During the past year, New York state has enacted legislation making mandatory the testing of all public school children by the audiometer. A considerably larger number of children with hearing loss has been revealed with the audiometer than with the older methods of testing. No test can be performed accurately when made in the presence of distracting or masking noises. Each ear must be tested separately.

Audiometric surveys of school children have revealed important facts. Retardation occurs at least twice as often among children with a hearing defect as among normal-hearing children. Speech defects occur eight times as frequently, and an inferiority complex and behavior problems are often traceable to neglected or uncompensated hearing deficiencies.

Another important problem in education of the hard-of-hearing pupil is the provision in the regular schools for instruction in lip reading and speech correction for those who need it to keep up with their classes. Children with severe loss of hearing or marked speech disturbances who cannot continue with the regular classes must be given this special help in residential or day schools, but should not be segregated with the totally deaf. Suitable electrical hearing devices for those who can benefit by their use should be provided. Another problem is the conservation of residual hearing. Even a slight remnant of hearing power in the speech range, when supplemented by lip reading or, in some cases, by a hearing aid, is useful in acquiring proper speech. Other problems are vocational guidance for older children and instruction in lip reading for adults. Prevention of deafness and the proper care of the hard of hearing are national educational and medical problems of large magnitude. The chief obstacles in the way of an early solution are, first, general lack of knowledge concerning the fundamental principles involved and the great possibilities of attainment through intelligent, organized effort and, second, a pathetic lack of funds. A well directed campaign and proper propaganda can overcome these difficulties.

19. Newhart, Horace: Hearing Problems in Education, J. A. M. A. **109**: 839 (Sept. 11) 1937.

Ciocco [20] reports on a statistical analysis of the variations in the hearing of 543 school children examined twice in a period of about three years. The same examiner made the tests, and the same type of audiometer was used. A fluctuation of 5 decibels above or below the original threshold was found in the greatest number of ears for tones up to 2,048 cycles, and a fluctuation of ±10 decibels, for tones of 4,096 and 8,192 cycles. The hearing of exactly two thirds of the ears remained relatively unchanged. The ears that varied for a particular tone varied in the same degree for other tones. There was a positive relationship between the variations in hearing in the right and those in the left ear. Ears with impaired hearing showed greater variability than those with good hearing. The ears with impaired perception for high tones, including that of 2,048 cycles, showed less variations in the second test than those in which hearing for these tones was not impaired. The results for ears which had reached a threshold hearing loss for both air and bone conduction of from 30 to 40 decibels did not fluctuate. When improvement in hearing occurred in the conductive type of deafness, it was found only in the ears with hearing the average threshold of which for all tones did not exceed from 30 to 40 decibels. The incidence of a definite decrease in the perception of high tones at the second test was significantly higher in boys than in girls.

Hunt [21] makes a plea for the establishment of clinics for the prevention of deafness. The plan was first advanced by Dr. Ross Faulkner nearly ten years ago and was put into operation on a smaller scale by Dr. Edmund Prince Fowler. The purpose is the establishment of many small preventive clinics in the schools, each of which works with a neighboring hospital otologic clinic. Printed information is to be given children and parents explaining the causes, early origin and means of detection of impaired hearing. The clinic should encourage attendance, adequate social service to look to the proper home life and environment, complete history taking and examination and subsequent treatment. The greatest hope in the prevention of deafness is in avoidance of involvement of the ear from infection. The running ear is the great offender.

Samuelson [22] states that there are three objectives in instruction on lip reading: (1) to help the student master an art, (2) to teach him to grasp thought and (3) to train the eye to see speech patterns as they are revealed by the organs of speech. In some methods of teaching the

20. Ciocco, Antonio: Audiometric Studies on School Children, Ann. Otol., Rhin. & Laryng. **46**:55 (March) 1937.

21. Hunt, Westley M.: The Role of the Deaf-Prevention Clinic, Laryngoscope **47**:241 (April) 1937.

22. Samuelson, Estelle E.: Fundamentals of Lip-Reading, Including Demonstrations with the Audience as Subjects, Laryngoscope **47**:237 (April) 1937.

student learns the movements by practice, under careful guidance. In other methods he is made conscious of the kinesthetic eye-training and mind-training processes. For example, he is taught that "th" is a tongue to teeth movement and "b" a movement with the lips shut. These movements are given in words and sentences until their recognition becomes subconscious. It is not necessary to see each syllable or word in a sentence. Lip reading is thought grasping. Lip reading is a substitute for hearing. It is a supplement, the eye and mind supplying what the ears do not hear. The majority of lip-reading teachers use voice in their lessons. It requires from sixty to one hundred and twenty hours of study to impart the fundamentals of teaching lip reading to prospective teachers.

Ballenger [23] studied further the effect of treatment with an audio-oscillator of special design. Twenty-seven partially deaf persons having various types of hearing impairment were treated, receiving a total of 1,797 treatments. The ascending and descending scales of tones were introduced into both ears, at a volume best tolerated by the patient. Treatments lasted thirty minutes and were given five times a week. Audiograms were taken before, at frequent intervals during and after the course of treatments. The averages taken for all the frequencies in all patients before and after treatments were identical to within one tenth ($\frac{1}{10}$) of a sensation unit. Ballenger concludes naturally that this form of therapy has proved of no value in the present investigation.

In 1919 the American Society for the Hard-of-Hearing was founded. The purpose of the organization is to serve as a center of information on problems of defective hearing, to improve the educational, economic and social conditions among both adults and children and to stimulate scientific efforts in the prevention of deafness and the conservation of hearing. Statistics gathered by Wright [24] for 1935-1936 showed that 805,435 children were tested in one hundred cities. Of these, 44,983 had impaired hearing; 22,216 were examined at clinics for diseases of the ear; 880 were restored to normal hearing, and 13,930 are now receiving instruction in lip reading. The program of this organization covers:

1. Prevention by means of hearing tests and treatment.

2. Education through instruction in lip reading in the regular schools and special classes for children with marked impairment.

3. Special training in speech and voice, when necessary.

23. Ballenger, Howard C.: The "Aural" or "Acoustic" Method of Treating Deafness: Further Investigations, Ann. Otol., Rhin. & Laryng. **45**:632 (Sept.) 1936.

24. Wright, Betty C.: National Organizations for Service to the Hard-of-Hearing: The American Society for the Hard-of-Hearing, Laryngoscope **47**:226 (April) 1937.

Weille and Billings [25] made a study of the efficiency of portable carbon microphone hearing aids, in order to determine how many decibels higher a patient's hearing curve is with the use of a hearing aid. This was done by determining the amplification curves for a variety of electrical carbon microphone hearing aids when in actual use by both normal and hard-of-hearing persons. In every experiment the rheostat was set at its maximum, and fresh batteries were always employed, together with an amplifier. The method used gave a measure of the value of the hearing aid under actual working conditions.

The curves obtained revealed a peak of amplification for the air conduction aids at 1024 d. v. This is the middle of the five octaves most important for speech perception, namely, from 256 to and including 4,096 cycles. There was absence of amplification of the high-pitched sounds. The amplification curves were not linear or uniform. As a result, some sounds would be perceived as sound and not as intelligible speech. From the curves obtained, it was obvious that persons with hearing at less than 1 meter would not be able to hear speech easily at a lecture or the theater with any of the hearing aids tested. The air conduction curves showed a maximum amplification value of only 20 decibels, or less. When the curve as a whole is considered, and not merely its peak, the amplification covers considerably less. By determining the minimum perceptible loudness differences, the authors found in this study that deaf patients utilize what hearing they have more keenly than normal persons. They suggest that further studies on the efficiency of hearing aids be made and that the data be made available to otologists. They also state that the selection of a hearing aid should be a service, and not a "sale," by the manufacturer.

Geffcken and Bayer [26] state that there are needed: (1) practical methods for testing physical qualifications for hearing aids, and (2) hearing aids adjusted to the individual need of the patient. It has been shown that such an instrument, although not fitted exactly, may be useful. The authors make plea for exact measurement of sounds from 200 to 6000 d. v. and intelligibility tests as guides to proper hearing aids. They promise to describe soon an instrument for measuring hearing aids that will be practical for the specialist.

Eschweiler [27] states that with poor contact between the ear and the ear piece low tones are not clear and high ones show too great intensity.

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25. Weille, Francis J., and Billings, Bruce H.: A Study of the Efficiency of Carbon Microphone Hearing Aids, New England J. Med. **216**:790 (May 6) 1937.

26. Geffcken, W., and Bayer, H. G. A.: Hörprüfung und Hörverbesserung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:335, 1937.

27. Eschweiler, H.: Absolute Eichung von Audiometern, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:190, 1936.

Meissner [28] states that he occludes one ear with the electrical ear olive of Langenbeck, without disturbing the other by air conduction. Testing with forks up to a-1 is practical. Sounds of higher pitch are carried over. If the difference in hearing in the two ears is over 60 decibels, the poorer ear can be tested only if an electrical noise apparatus is used.

In the modified test described by Coldstein [29] the stem of the fork is set on the manubrium sterni. The sound is referred to the hand corresponding to the side of conduction deafness. The test may be successful when not lateralized from the vertex.

On making tests with the monochord in the negative pressure chamber, Hartmann [30] finds that at from 4 to 5,000 feet there is definite loss for high tones. The test for high tones may be used to determine early the probability of altitude sickness. Variations occur according to resistance, age, etc.

With use of a holder with a spring hammer and a fork of known decrement, and with a similar arrangement for the sounding rods for tones over 4000 d. v., Struycken [31] claims more accurate results than with an audiometer. The same instrumentarium may be used to test hearing aids.

Chklar and Drunnow report that in 100 cases of otosclerosis the manometer was inserted into the external auditory meatus and the ear inflated according to the method of Politzer. At the moment of insufflation the membrana tympani bulges and produces rapid agitation of the drops (*gouttes*) in the manometer. The authors noted the slow return of the drops (*retrait ralenti*) in 86 of 100 cases. The phenomenon is rare in other forms of deafness. (The manometer was not described. Perhaps this type measured by drop displacement instead of by the height of the column.)

Vestn [33] reports on seven months' observation in 126 cases of deafness. The early symptoms were: loss of hearing for forks c-4 and c-5 and shortened bone conduction time, but retention of hearing for whis-

28. Meissner, H.: Methoden der Hörprüfung eines Ohres, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:415, 1936.

29. Coldstein, M. A.: Modification of the Weber Test, Zhur. ushn., nos. i. gorl. bol. **13**:307, 1936.

30. Hartmann, H.: Obere Hörgrenze bei Sauerstoffmangel, Luftfahrt- med. abhandl. **1**:192, 1936.

31. Struycken, H. J. L.: Bestimmung des Minimum Audible mittelst Stimmgabeln und Klangstäbchen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **40**:483, 1937.

32. Footnote deleted by the author.

33. Vestn, Minkin E.: Zur Frage einer frühen Diagnostik durch Lärm verursachten Störungen des Gehörorgans, Zentralbl. f. Haut- u. Geschlechtskr. **28**:677, 1937.

per. Later, there was loss for both low tones and whisper. In the first stage recovery is possible if noisy occupation is abandoned.

Jannulis and Delijannis [34] report 29 cases of diabetes associated with loss for high tones, slight loss for low tones and normal or shortened conduction time in the Rinne and Weber tests on the good ear. The onset was sudden or gradual. In case of the latter the deafness is reversible. There were the same findings in cases of gout. The lesion is believed to be peripheral and toxic.

Schwarz [35] says that hereditary deafness can be judged only by careful study of the family tree. Collateral members must be included, to differentiate from the sporadic form. Lack of speech indicates either early onset or hereditary deafness. The sporadic form is more complete.

Bernovitz [36] states that blind persons determine the direction of sound better than seeing persons, probably owing to practice. Their eyes turn in the direction of the source of the sound, even in the presence of nystagmus.

Mittermaier [37] states that testing of bone conduction by a telephone instrument is not yet perfected and that the otoaudion can replace the continuous tuning fork series in routine tests for hearing.

Sangiovanni,[38] in examination of 100 pregnant women, found that irritability of the vestibule was present during the early months. There were diminution of hearing for both upper and lower tones, shortening of bone conduction time and slight changes in the membrana tympani. These disappeared after birth and were not related to the blood pressure.

Dederding,[39] in a paper read at a meeting of the Austrian Oto-Laryngological Society at Innsbruck, Austria, May 28, 1937, arrives at several conclusions. In her functional testing of hearing she does not use an audiometer because tuning fork methods have been employed for a long time so that their various advantages or disadvantages are well known, whereas the qualities of the audiometer have not yet been

34. Jannulis, G., and Delijannis, G.: Diabetes und Gicht als Ursache von Kochlearisschädigung, Monatschr. f. Ohrenh. **70**:1504 (Dec.) 1936.

35. Schwarz, Max: Familiennachweis und Organbefund bei der Begründung der erblichen Taubheit, Erbbl. f. d. Hals-, Nasen- u. Ohrenarzt **28**:15 (Feb.) 1937.

36. Bernovitz, M.: Ueber das Richtungshören der Blinden, Monatschr. f. Ohrenh. **70**:984 (Aug.) 1936.

37. Mittermaier, R.: Ueber audiometrische Höruntersuchungen der Knochenleitung, Hals-, Nasen- u. Ohrenarzt **27**:201, 1936.

38. Sangiovanni, V.: Sulla funzionalità uditiva in gravidanza, Arch. ital. di otol. **48**:217 (April) 1936.

39. Dederding, Dida: Die Bedeutung der bisher üblichen Methoden der akustischen Funktionsprüfung für die Diagnostik mit besonderem Hinblick auf die Bestimmung der oberen Hörgrenze, Monatschr. f. Ohrenh. **71**:1045 (Sept.) 1937.

fully determined. Furthermore, she believes that in the majority of aural conditions hearing for the lower tones comes most into consideration. It is for the two lowest octaves that the audiometer is more or less undependable. Furthermore, it is her opinion that the audiometer gives uncertain findings in the highest octaves, where the signs of impairment in perception first manifest themselves. For these reasons, and because Bezold through tuning fork tests established facts which later generations have thus far been able to change or to improve only slightly, Dedering is unwilling to give up the regular tuning fork tests.

Fowler [40] presents further observations on the phenomenon of the recruitment of loudness with intensity, on which he first reported in 1928 at the meeting of the American Otological Society. He calls attention to the limitation of the tests commonly used in diagnosis and states that any or all of the standard tests may appear to give positive results for nerve deafness which may not be present and that their diagnostic significance is based on bone conduction, which is often unreliable. The test he again describes enables one to differentiate nerve from conduction deafness without measuring air or bone conduction thresholds and, except in certain instances, without using bone conduction in any form. It is the only reliable test for nerve deafness which does not require confirmation by other tests.

The test consists of measurement of the recruitment of loudness with intensity by balancing the loudness of sounds thrown alternately into one ear and then into the other. The differences in loudness necessary for balance at the various steps above threshold measure the rates or recruitment of the neural elements necessary for equal balance. This gives one a measure of the location, number and extent of loss of the neural elements which initiate, conduct or relay impulses from the end-organs to the cortex. The fundamental observations reported in a previous article are as follows: In monaural or unequal binaural pure conduction deafness, there is no change at any intensity in the relative sensations of loudness in the two ears. If the difference in hearing at threshold is 30 decibels, it will remain at 30 decibels for all intensities above threshold. In other words, 30 decibels will have to be added to the poorer ear to make the sound appear as loud to it as to the better or normal ear. In monaural or unequal binaural nerve deafness, the poorer-hearing ear hears more and more acutely as the intensity of sound increases. For example, if the difference between the two ears is even as much as 75 decibels at threshold, there may be little or no difference at an intensity well above threshold. These phenomena occur in varying

40. Fowler, Edmund Prince: The Diagnosis of Diseases of the Neural Mechanism of Hearing by the Aid of Sounds Well Above Threshold, Laryngoscope **47**:289 (May) 1937.

degrees at different frequencies and vary with certain types of curves. Fowler presents graphs and cases showing several apparent exceptions and precautions which one should take, such as the possibility of over-masking.

There is a difference in the quality of the sound heard by the impaired or poorer ear. In nerve deafness the sounds are cloudy and often seem to differ in pitch, but at threshold are suddenly distinct. In conduction deafness sounds just above threshold appear and disappear and are indistinct; the patient is in doubt as to whether they are heard or not, and often as to whether they are felt. Fowler also shows how this test is applicable even when hearing curves are the same for the two ears. He shows a series of graphs based on observations in actual cases which demonstrate how at any frequency the "alternate binaural loudness balance" measures the recruitment of loudness in cases of deafness due to various combinations of lesions of the middle and inner ear. There is an invariable recruitment phenomenon in every case in which nerve deafness is indicated by loss of bone conduction. He calls attention to the phenomenon the significance of which is obscure, in which the ear deafened by a nerve lesion may hear better than the opposite, better ear. His data also show that less intense masking is required for an ear with nerve deafness than for one with conduction impairment. Whenever an ear is easily masked it is either normal or affected by a nerve lesion. Nerve deafness may be masked by faint tinnitus at or below the testing frequency, and the true threshold may be observed only in the absence of tinnitus. The ratio of loudness recruitment furnishes a means for estimating the masking effect of tinnitus.

A malingerer or person with hysterical deafness cannot reproduce the loudness balance tests. The recruitment phenomenon may also be applied to study of the relative intensity of sensations from other sense organs.

Crowe [41] reports his findings on patients with Ménière's and pseudo-Ménière's disease. He has examined 175 patients in whom Dr. Dandy divided the auditory nerve or the vestibular portion of the nerve, for relief from vertigo. Otoscopic, tuning fork and audiometric tests were made on all these patients before and after operation. He found that a patient with Ménière's disease has the classic signs of an organic nerve lesion. Repeated examinations have shown that the hearing acuity may fluctuate. This variation is also true for bone conduction. He concludes that in cases of Ménière's disease even total absence of bone conduction on one or both sides does not necessarily indicate irreparable

41. Crowe, S. J.: The Diagnosis of Nerve Deafness, Laryngoscope **47**:492 (July) 1937.

damage to the inner ear or nerve. The observations on tinnitus disclose several facts: First, tinnitus is not the cause but a symptom of the lesion, producing impairment of hearing by bone and by air. Furthermore, unilateral tinnitus does not mask hearing by bone conduction in the opposite ear. Functional tests and observations at operation on patients with Ménière's symptom complex have convinced observers that the lesion is probably in the nerve or the auditory pathways in the brain and is not due to pressure changes in the cerebrospinal or the intracochlear fluid. Crowe stresses the importance for the advancement of knowledge of Ménière's disease, as well as for a better understanding of the less severe, but common, disorders of the auditory and vestibular apparatus, that postmortem examination be made in all cases in which the patient has been accurately tested and that these studies include the central auditory and vestibular pathways. He also brings out the necessity for masking for accurate testing for either bone or air conduction acuity in patients with normal hearing in one ear and partial or total deafness in the other.

Macfarlan [42] states that one can judge an otologist as to his ability to test hearing by his familiarity with the methods for detecting errors in the patient's reporting. When there are great variations in the tests a number of factors may be at fault. A varying noise level may be changing the damping of the sound. A varying technic may change the intensity of vibration. The patient may be exhibiting tone memory, and in testing children, attention is a great factor. Each of these items must be covered for an accurate report. Many methods of damping can be used to invent a great variety of tests for malingering. The ordinary voice test, the use of Bárány's noise apparatus, the Stenger test and the plugged stethoscope test are mentioned for the detection of so-called malingering.

The use of timing with tuning forks after the duration of vibration for the fork is known is a valuable test for simulated deafness. Modification of the Weber test by placing the finger in the tested ear after the end-point for bone conduction has been reached, which usually brings back the sound, is a good test. The unilateral vestibular tests are valuable for confirmatory evidence. Macfarlan suggests that parents who have a young deaf child to be tested before speech has been developed should condition their child to respond by raising the hand to sounds loud enough to reach the tactile sense. When the otologist tests this child, he may drop the intensity below the tactile threshold, and if the child responds correctly he must be reporting hearing.

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42. Macfarlan, Douglas: Simulated Deafness (Malingering), Laryngoscope **47**:538 (Aug.) 1937.

Ciocco [43] reports his findings, as well as those of other men, with reference to investigations on hearing in syphilitic patients. He finds that syphilis apparently does not cause impairment of the auditory nerve more often than any other general systemic disease. There is no particular type of nerve deafness which is found more frequently than others in syphilitic patients. However, when hearing impairment does occur, it is found more frequently in persons with neurosyphilis. Antisyphilitic treatment is neither beneficial nor deleterious in the majority of cases.

Dr. Alfred Lewy, Chicago, assisted in the preparation of this summary.

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43. Ciocco, Antonio: Nerve Deafness from Syphilis, Laryngoscope **47**:572 (Aug.) 1937.

Correspondence

PERFORMANCE CHARACTERISTICS OF ELECTRICAL HEARING AIDS FOR THE DEAF

To the Editor:—In our paper which appeared in the June 1936 issue of the ARCHIVES (Performance Characteristics of Electrical Hearing Aids for the Deaf, ARCH. OTOLARYNG. **23**:617, 1936) we gave the results of extensive tests which we made of four nationally advertised portable hearing aids of the carbon microphone type equipped with both air and bone conduction receivers. We carefully protected the manufacturers, who generously cooperated with us, from any possible unfavorable comparison of the merits of their respective products by designating by number the instruments tested, keeping their identity unrevealed.

In the advertising pages of the September 1937 issue and of several previous issues of the *Revue de l'Ouie,* a bimonthly organ of two national organizations for the hard of hearing in France, there appeared in the advertisement of the Companie Française des Microphones Acousticon, the French subsidiary of the Dictograph Products Company, Inc., of New York, the following statement:

"The Medical Association of the United States [referring to the American Medical Association] has accepted the Acousticon instruments.

"In the 'Archives of Otolaryngolgie' (June 1936) Drs. Hartig [misspelled Hartyr] and Newhart relate the experiments they have made with electro-acoustic instruments of four different makes and after the results were obtained through the aid of these instruments, *Acousticon came out ahead under Number 1. These doctors consider the Acousticon as the most perfect instrument from every point of view.*"

We emphatically deny having made any statement of this nature and declare the unwarranted use of our names to promote sales of the products of the company mentioned to be untruthful, unethical and injurious to our reputation among our scientific and medical colleagues.

The vice president of the Dictograph Products Company, Inc., Mr. D. D. Halpin, has apologized to us for the misuse of our names and denies all responsibility in the matter.

HENRY E. HARTIG, PH.D., and HORACE NEWHART, M.D., Minneapolis.

Abstracts from Current Literature

Ear

Cranial Venous Sinuses: Correlation Between Skull Markings and Roentgenograms of the Occipital Bone. Barnes Woodhall and Asa E. Seeds, Arch. Surg. **33**:867 (Nov.) 1936.

A preliminary study of 100 consecutive routine roentgenograms of the occipital bone of normal persons, taken in the anteroposterior projection plane, has demonstrated relative differences and variations in the size of the bony markings of the lateral sinuses that correspond closely to the anatomic observations previously reported. The normal standard of variation, appraised by roentgenographic study, is of clinical value in determining the significance of irregularities obtained by the Queckenstedt test and the dynamics of the venous return from the brain in the presence of thrombosis of the lateral sinus or the jugular vein.

Grant, Philadelphia. [Arch. Neurol. & Psychiat.]

Indications for the Labyrinth Operation with Especial Reference to Acute Diffuse Destructive Labyrinthitis. R. Lund, J. Laryng. & Otol. **51**:425 (July) 1936.

Lund deals exclusively with nonspecific inflammatory labyrinthine disease which arises from the middle ear in persons with acute or chronic aural suppuration. He differentiates chronic diffuse destructive labyrinthitis, circumscribed labyrinthitis, serous labyrinthitis and acute diffuse destructive labyrinthitis. He gives details of the various types of treatment of diffuse destructive labyrinthitis which he has used in the last twenty-eight years. For six years he adhered to the radical view of the Vienna School which maintains that every case of acute labyrinthitis in connection with a dead labyrinth indicates the necessity for immediate labyrinthectomy; then for eight years he adhered to a conservative view, no labyrinthectomy being done unless there were clinical signs of meningitis, and finally, in the last period of fourteen years, his indications for labyrinthectomy were made dependent on the result of examination of the cerebrospinal fluid. In the last period the best results have been obtained.

Editor's Abstract.

Test for Functional Aphonia and for the Detection of Complete Unilateral Feigned Nerve Deafness. N. Asherson, J. Laryng. & Otol. **51**:527 (Aug.) 1936.

According to Asherson, speech is heard by air or by bone conduction. A person speaking is heard by another by air conduction. He hears his own voice by bone conduction and reflexly modulates its intensity by his own perception of it. If the acuity of autoperception (by bone) is increased or diminished, the tone of the voice is correspondingly adjusted; e. g., the person with advanced bilateral nerve deafness (a lesion in which the bone conduction is diminished) speaks in a loud tone. If a person with normal hearing is temporarily put in a position in which he is unable to hear himself speak, he is in the position of the person with complete nerve deafness. This may be accomplished by "occupying" the ear by an extraneous noise, in one of two ways. 1. Two Bárány noise boxes are required. The nozzle of the activated noise box is inserted into each ear. 2. In the absence of a noise box, a sheet of firm writing paper is pressed loosely against each ear by an observer standing behind the patient. The paper is continuously and rapidly oscillated to and fro. The resulting noise will effectively "occupy" the ear. If a person with normal hearing is instructed to read aloud

continuously while both his ears are "occupied" by a source of noise as described previously (i. e., he is now unable to hear his own voice), it will be found that he immediately raises his voice considerably. This raised voice is maintained all the time his ears are deafened. The patient is unaware of the fact that he has raised his voice, which immediately reverts to the normal when the extraneous noise ceases. This phenomenon may be turned to clinical use in the detection and treatment of functional aphonia, and it may also be applied to the detection of complete unilateral feigned nerve deafness. The author gives the details of these tests.

EDITOR'S ABSTRACT.

MULTIPLE APICAL EMPYEMA OF THE PETROSA WITH MULTIPLE PERFORATIONS INTO THE CRANIAL CAVITY AND INTO THE INTERNAL AUDITORY CANAL. H. RICHTER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141**:334, 1937.

Richter shows that empyema originating in the pneumatic cells of the petrosa is more dangerous than empyema of the mastoid that develops after otitis media. This greater danger is due to the fact that the petrosa is more difficult to approach. The author reports the clinical history of a man, aged 66, in whom mucous otitis of five weeks' duration resulted in fatal suppurating meningitis. Operation on the mastoid of the patient, who was hospitalized in a state of unconsciousness, did not disclose empyema and did not clarify the mode of transmission of the aural suppuration to the meninges. However, the histologic examination of the petrosa revealed that the pneumatization of the diseased petrosa was well developed. After describing the extension of the pneumatization, the author points out that the entire cell system was, as is usual, involved in the inflammatory process. Corresponding to the duration of the otitis media, the bony structure of many cells and of the adjoining medullary spaces shows considerable change. There are three characteristic sites at which larger empyemas exist, but there are also smaller empyemas. The three larger empyemas in this case caused several perforations, among them three endocranial ones. The retrolabyrinthine empyema perforated the dura of the posterior cranial fossa. The empyema on the upper surface of the petrosa penetrated the dura at two sites: it caused perforation of pus into the middle cranial fossa and suppurating infiltration of the internal auditory canal. Thus the fatal meningitis may have developed simultaneously by several routes. The author emphasizes that the extensive pneumatization of the petrosa was the basis for the development of the empyema in the petrosal apex.

EDITOR'S ABSTRACT.

ABOLISHMENT OF VESTIBULAR FUNCTION BY REFLEX ACTION. F. ALTMANN, Monatschr. f. Ohrenh. **70**:709 (June) 1936.

Altmann directs attention to the fact that in some cases the destruction of one labyrinth is accompanied by impairment of the vestibular irritability of the other. After reviewing the literature, he describes a case of his own observation, in which labyrinthitis with deafness and abolishment of the vestibular irritability developed on the left side. Examination of the right ear revealed an almost normal capacity for hearing but complete unresponsiveness to caloric stimuli and considerable reduction of the reactivity to stimulation by rotation. Gradually the responsiveness to vestibular stimuli returned, the return being much more rapid on the right than on the left side. The author expresses the belief that the temporary functional exclusion of the contralateral labyrinth is due to reflex processes elicited in the center of the diseased labyrinth.

EDITOR'S ABSTRACT.

SYMMETRY OF RESIDUAL HEARING AND HEREDITY. B. LANGENBECK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:486, 1936.

Langenbeck takes up once more his so-called symmetry law of hereditary deafness. He admits that medical diagnosis can reckon to a large extent on probabilities only and that the diagnosis of heredity is no exception to this rule. He

points out that in his diagnoses of "definitely hereditary" deafness, deafness in other siblings was always a requirement, except in case of an only child, and when blood relationship of the parents and the presence of other hereditary defects formed the basis for his diagnosis. He again reaches the conclusion that the law of symmetry of residual hearing is of practical value in determining the hereditary nature of deafness, provided of course it is employed with critical reserve.

EDITOR'S ABSTRACT.

TREATMENT OF INFLAMMATORY CONDITIONS OF THE EXTERNAL AUDITORY CANAL WITH BRILLIANT GREEN. I. T. DOROSHENKO, Vrach. delo **18**:479, 1936.

Doroshenko points out the efficacy of brilliant green in the treatment of inflammatory conditions of the external auditory canal of the child.

Brilliant green exerts a marked bactericidal action, particularly on the staphylococci and is therefore of great value in the treatment of furunculosis and other inflammatory conditions of the external auditory canal, which occur so frequently in children after bathing, as well as other conditions in which maceration of the epithelium occurs.

Brilliant green is used in a 1 per cent solution in 40 per cent alcohol or in the form of an ointment. A tampon saturated with the ointment or with the solution of brilliant green is introduced into the external auditory canal and left there for a day.

This method of treatment was used on 106 patients from the ages of 1 to 16 years. The maximum duration of the infection was three days in 15 per cent of these patients, while in the remainder the duration was even less. The author strongly advocates this method of treatment.

BODER, Los Angeles. [AM. J. DIS. CHILD.]

Pharynx

ACUTE STREPTOCOCCAL INFECTION OF THE THROAT. E. D. D. DAVIS, Brit. M. J. **2**:617 (Sept. 26) 1936.

This is the second paper in the symposium. Davis gives the various names under which this condition has been known. The subdivisions depend on degree. The first is tonsillitis, with much variation in the extension of the inflammation and edema to the surrounding tissues. The second is gangrenous inflammation, with a sloughing membranous exudate and marked toxemia. This may begin as acute tonsillitis and extend to the more severe condition. Under symptomatology Davis mentions the fulminant conditions, accompanied by edema involving the epiglottis and arytenoid cartilages, with difficult swallowing, even inability to swallow saliva. Primary streptococcic laryngitis is rare; cervical adenitis is always present. Ludwig's angina may be a complication and may lead to suppuration. He thinks that if tonsils have been removed previously, both the inflammation of the pharynx and the adenitis are more marked, but both may appear even when the tonsils have not been removed. The most common cause is Streptococcus haemolyticus; other organisms may complicate the picture. Even a pneumococcus alone is sometimes found as a complication. The causal factors in epidemics are discussed. Under treatment he discusses careful, skilful nursing. Careful bacteriologic study, blood transfusions and immunizing blood transfusions may be necessary. Antiscarlatinal streptococcic serum should be given in large doses in streptococcic infections. Antidiphtheritic serum may be of value even when the Klebs-Löffler bacillus is absent and is never of value in the presence of a pneumococcus. Neither does the antistreptococcic serum do any good when there is pneumococcic involvement. Suitable types of pneumococcus serum may be used. Davis stresses purgation with mild mercurous chloride; gargles are of little value. Swabbing with hydrogen peroxide and then spraying with a mild antiseptic are thought to be good. Sleep must be induced with powder of ipecac and opium or some other

hypnotic. Hot applications are made to the neck in the form of electric heating and inhalation of steam. Potassium chlorate has its value. Fluids administered by mouth and dextrose injected intravenously are of undoubted benefit. Complications should be treated as they arise. Tonsillectomy should not be performed during the acute stage. When hemorrhage results from a tonsillar abscess, tonsillectomy may be necessary. Cervical adenitis should not be treated by incision early. Tracheotomy is indicated in edema of the glottis.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

TONSILLECTOMY FOR TONSILLAR ABSCESS. A. LINCK, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141**:255, 1936.

According to Linck, it is now generally recognized that in the treatment of paratonsillar abscesses tonsillectomy is helpful and necessary. That tonsillectomy during the acute stage does not involve the dangers formerly ascribed to it is likewise no longer questioned. However, about the time and the general or selected application of tonsillectomy opinion is still divided. The majority of clinicians and practitioners still advise incision during the acute stage and recommend tonsillectomy only as a secondary operation. Tonsillectomy without previous incision is considered justified only in cases in which there are complications or especially severe involvement. The author, however, is of the opinion that abscess tonsillectomy, with complete exclusion of incision, should be performed as the primary intervention in all cases in which a paratonsillar abscess either is clinically demonstrable or appears probable. He regards incision in addition to tonsillectomy, within the complete therapeutic program, as entirely superfluous and often harmful. Moreover, he does not think that a comparison is justified between incision and abscess tonsillectomy, for incision is only a partial treatment, with limited therapeutic aims and possibilities, whereas abscess tonsillectomy represents a complete treatment, with the aim of complete and final arrest of the process. The author estimates the two treatments from three different standpoints: the risk involved in the abscess, the results of the two operations and the risk involved in the two operations. He reaches the conclusion that there is only one factor that could be cited against the general performance of abscess tonsillectomy, namely, the conviction that the tonsils are indispensable to the patient and that they should be preserved even in cases of paratonsillar abscess. This, however, contradicts the accepted opinion, which he regards as definitely established.

EDITOR'S ABSTRACT.

DIAGNOSIS AND THERAPY OF SUPPURATIONS OF THE MOUTH AND THE PHARYNX ASCENDING TO THE ENDOCRANIUM. B. KECHT, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141**:275, 1936.

Kecht points out that generalized complications following acute suppurations of the oral and pharyngeal cavities originate in the teeth, the tonsils, the pharyngeal wall or the retropharyngeal space. He reports two cases of dentogenic complications, four cases of tonsillogenic complications and one case of pharyngogenic complications. In discussing the dentogenic complications, he says that in one case there existed sanious leptomeningitis and sanious thrombophlebitis of the right cavernous sinus and septicopyemia; in the second case there were thrombophlebitis of the cavernous sinus and septicopyemia. The tonsillogenic complications consisted: in one case, of basal leptomeningitis and suppurating thrombophlebitis of both cavernous sinuses and of the right sigmoid sinus; in a second case, of suppurating pachymeningitis interna and leptomeningitis, abscess in the cortex of the left temporal lobe and suppurating thrombophlebitis of both cavernous sinuses; in a third case, of pachymeningitis interna, leptomeningitis and encephalitis; in a fourth case, of leptomeningitis and thrombosis of the right cavernous sinus. In the case of pharyngogenic complications, there existed leptomeningitis with hydrocephalus and sepsis. In discussing the pathogenesis of these complications, the author points out that the transmission from the main focus may take place (1) by

way of the loose tissue of the parapharyngeal space upward to the cranial base in the form of abscesses or phlegmons and by secondary passage through the various foramina or (2) by venous passages, namely (*a*) by way of the pterygoid plexus, whence the passage goes further by way of the venous connections into the cranium or (*b*) by retrograde spreading of thrombotic material from the posterior facial vein by way of the internal jugular vein and the sigmoid sinus or the carotid plexus. Most frequent is the transmission by a combination of the passages mentioned under points 1 and 2*a*. The author classifies the symptoms into extracranial and intracranial ones. As the most frequent extracranial symptoms he mentions rigidity of the jaws, toothache (trigeminal neuralgia), parotitis, swelling of the temples, aural symptoms (piercing pains, tinnitus, impairment of hearing), piercing pains in the eyes, homolateral chemosis and protrusion. The intracranial symptoms are trigeminal neuralgia, eventually with herpes zoster, contralateral or bilateral ocular symptoms, and manifestations such as vomiting, headaches and pathologic changes in the spinal fluid. The course of the process varies, but most frequently it is foudroyant, terminating fatally within a few days. However, the prognosis is not necessarily unfavorable, for if the disorder is recognized early and treated properly, cure is possible. In the treatment it is essential to remove the primary focus (through tonsillectomy, extraction of a tooth or incision of a retropharyngeal abscess), to open the parapharyngeal space and the fossa pterygomaxillaris, to make the necessary orbital operations and to inspect carefully and perhaps ligate the large veins.

EDITOR'S ABSTRACT.

HYPERTROPHY OF TONSILS AND BACTERIAL DISEASES. S. PELLER, Monatschr. f. Ohrenh. **70**:851 (July) 1936.

Peller shows the numerical relationship between hypertrophy of the tonsils in young persons and the incidence of such diseases as scarlet fever, diphtheria, nephritis, rheumatism, otitis media and appendicitis. Anamnestic investigations revealed to him that young persons with hypertrophy of the palatine tonsils have scarlet fever and diphtheria slightly more often and nephritis and appendicitis much more often than do young persons whose tonsils are not enlarged. Rheumatism and otitis show the same incidence in those with and those without hypertrophy of the palatine tonsils.

EDITOR'S ABSTRACT.

Nose

ETHMOIDAL SUPPURATION WITH ORBITAL INVOLVEMENT. D. PATERSON, Proc. Roy. Soc. Med. **29**:1435 (Sept.) 1936.

Five weeks before the patient, a girl of 2 years, was admitted to the hospital she had contracted measles, and she had been listless and in poor health since. A week before admission she had become acutely ill with severe pain in her forehead and attacks of shivering. About the fourth day of the illness, puffiness and redness of the right upper eyelid occurred. The next day the eyelid became markedly swollen, and the palpebral fissure was obliterated. The clinical examination, the progress before operation, the operation, the progress after operation and the investigations are given in detail.

WILLIAMSON, New Orleans. [AM. J. DIS. CHILD.]

UNUSUALLY LARGE MUCOCELE OF THE FRONTAL SINUS, WITH SEVERE VISUAL DISTURBANCES AND RECOVERY. F. CSILLAG, Klin. Monatsbl. f. Augenh. **97**: 663 (Nov.) 1936.

The right eye of a woman aged 52 was swollen for about five months prior to observation by the author. The attending general practitioner had tried to empty the tumor by insertion of a cannula into the upper lid. Preceding grip, syphilis and tuberculosis were excluded. The right palpebral fissure was narrowed and displaced downward and temporally; the upper lid showed ptosis and was

immovable, cyanotic and crossed by distended blood vessels. No tenderness on touch existed. A massive, tight but barely fluctuating tumor the size of a walnut was seen below the upper lid. It arose from the frontal bone, causing exophthalmos. The upper orbital margin was not palpable. The eyeball was flattened from above. The disk was edematous and prominent; the retinal veins were slightly distended and tortuous, and retinal hemorrhages were absent.

A roentgenogram revealed the upper margin of the orbit greatly deviated upward and the entrance of the orbit and the frontal sinus generally widened, while the periosteum of the temporal upper margin was thickened. An incision was made along the eyebrow; the lid was pulled downward, and the tumor was removed; it burst during extirpation. The mucocele extended backward toward the optic foramen, diverting the optic nerve; the osseous wall of the orbit and an area of the dura mater were eroded. Nevertheless, prompt recovery followed, and vision of 5/10 of normal was regained, with a prospect of further improvement.

K. L. Stoll, Cincinnati. [Arch. Ophth.]

Roentgenography and Conservative Therapy of Accessory Nasal Sinuses by Means of Displacement Method of Proetz and Le Mée. A. Laskiewicz, Monatschr. f. Ohrenh. **70**:834 (July) 1936.

Laskiewicz directs attention to the auto-instillation method (according to Proetz and Le Mée) by which contrast mediums are introduced which have a therapeutic effect. After reviewing the development of this method, the author stresses the factors that are important in the application, such as that the rarefaction of air should be done with an electrical suction pump, that the negative pressure should never exceed 180 mm. of mercury, that not more than 4 or 5 cc. of iodized poppy-seed oil should be instilled into each side of the nose and so on. He describes a number of cases in which he employed the instillation method.

Editor's Abstract.

Cure of Chronic Suppuration of Frontal Sinus Without Disfigurement. O. Muck, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:477, 1936.

The serious disadvantages of the radical operation on the frontal sinus, namely, the disfigurement, the postoperative supra-orbital neuralgia, the occurrence of double images and often the persistence of the suppuration, induced Muck to resort to another method, a combination of surgical and physical therapy. It aims to preserve the bony wall of the frontal sinus so that disfigurement and supra-orbital neuralgia are avoided. The swollen and edematous condition of the mucous membrane is counteracted by the action of tincture of iodine. The author's procedure is as follows: With general or local anesthesia an incision about 2 cm. in length is made at the root of the nose. Then a circular opening about 1 cm. in diameter is chiseled through the anterior wall of the frontal sinus. The mucous membrane under this opening is carefully removed, and the pus is removed from the frontal sinus by means of a solution of sodium chloride. After this solution has been removed, a 10 per cent solution of iodine in alcohol is introduced into the sinus. The opening is closed with a compress, and the head is moved so that the iodine-alcohol solution penetrates everywhere. Then the tincture of iodine is removed and the opening is closed by means of a rubber stopper that has two perforations; care is taken that the stopper does not touch the mucous membrane of the posterior wall of the sinus. Through the openings in the stopper the sinus is once more irrigated with solution of sodium chloride. The stopper remains in situ, and before a bandage is put on a sieve is placed over the sinus opening. The sieve insures access of air. After two days the irrigation is repeated. In the subsequent irrigations the solution of sodium chloride becomes clear, and when, after a week or later, the stopper is removed it can be seen that the swelling has been considerably reduced. Finally the wound in the soft parts is closed by clamp suture. The author states that with this treatment he effected cure in eight cases of chronic suppuration of the frontal sinus.

Editor's Abstract.

Miscellaneous

STAPHYLOCOCCIC IMMUNITY. J. S. KITCHING and L. N. FARRELL, Am. J. Hyg. **24**:268, 1936.

Active immunization of rabbits and mice with staphylococcus toxoid produced an increase in resistance against the lethal effect of staphylococci and staphylotoxin introduced parenterally and also against the dermonecrotic effect of staphylotoxin introduced intradermally in rabbits. In general, the protection against these two effects depended on the amount of circulating antitoxin as determined by serum titrations, the "standard unit" being used as a basis for comparison. In rabbits the titer of circulating antitoxin fell off approximately ten days after the last of a course of injections of toxoid. The drop was at first sharp, to approximately half, then gradual. The titer could be raised approximately to its former maximum by one further injection of toxoid. Treatment of rabbits and mice with formaldehydized staphylococcus vaccine, prepared and used under the conditions described, produced little or no circulating antitoxin and no increase in resistance against the lethal effect of staphylococci and staphylotoxin introduced parenterally or against the dermonecrotic effect of staphylotoxin in rabbits. The serum obtained on injecting formaldehydized staphylococcus vaccine into rabbits, in the quantity used, did not confer passive immunity against the lethal effect of staphylococci in rabbits and mice. Staphylococcic antitoxin was found definitely to confer passive immunity against the lethal effect of living staphylococci and staphylotoxin introduced parenterally into rabbits and mice. The immunity was in direct proportion to the number of units of antitoxin given. Antitoxin given intravenously before the injection of toxin was found to have a modifying effect on the toxin damage to tissues and, under the conditions of the experiment, definitely to prevent the lethal effect of the toxin or of the micro-organisms. Delay in administration of antitoxin, particularly when the subcutaneous route was used, distinctly lessened its value.

FROM THE AUTHORS' SUMMARY. [ARCH. PATH.]

TREATMENT OF PAROXYSMAL SNEEZING. F. COKE, J. Laryng. & Otol. **51**:522 (Aug.) 1936.

Coke classifies paroxysmal sneezing into allergic and microbic types and a type which is primarily of vasomotor origin. He shows that, while it is convenient to deal with the conditions as groups or types, it must never be forgotten that two or more factors may be at work in any one case. Of the allergic types of paroxysmal sneezing, he first discusses hay fever, pointing out that every person with hay fever responds with a large wheal when pollen is applied to a scratch on the skin. From other allergic causes the paroxysms of sneezing may occur all the year round, as from face powder or house dust; only in certain places, as in a house in which there are cats or dogs or in the proximity of stables, or under certain conditions, as while one is wearing certain furs. Sufficient information may be available to identify the protein at fault, but usually routine testing for dermal reactions is necessary. Not infrequently, foods may cause the attacks. The method of avoidance is effective if the protein is some rare food or fur. Face powders free from orris are available; animals and featherbeds can be dispensed with. Specific desensitization is done in that extracts of grass pollens, house dust and the appropriate animal hairs are injected in doses of increasing size. In a case of hay fever, attempts should be made to reach a large dose, of about 100 mg., of pollen before the season begins. With the other proteins, when a dose has been reached that controls the symptoms the intervals between the doses can be extended gradually up to an interval of two months. Twelve subsequent doses given at this interval usually cure the patient. About the nonspecific methods, the author says that in his opinion the best is the Danysz, or mixed coliform, vaccine, preferably autogenous. He has been able to relieve all symptoms in several cases of hay fever with these vaccines. Under the heading of microbic factors in paroxysmal sneezing the author discusses first the common

cold and then chronic infections. In the latter cases he found that it is always worth while to have the full pathogen selective test carried out. This means that all the organisms found to be present in the body are isolated and incubated for twenty-four hours in the patient's own blood. Any that survive may be judged to be those against which the patient's blood has no resistance (Crowe's technic). With small doses of such vaccines the patient may be cured of the paroxysmal sneezing. Regarding paroxysmal sneezing of vasomotor origin, the author says that this type is often seen in plump women between the ages of 20 and 50. They are prone to urticarial and asthmatic manifestations, and their blood pressure is exceptionally low. They complain of a "blown up" feeling after meals. The menstrual periods tend to be scanty and painful, and the breasts often show painful engorgement just before the menstrual flow takes place. Most of these symptoms, especially the swelling of the breasts, can be completely controlled with a few intravenous injections of colloidal manganese, in 20 minim (1.2 cc.) doses. A course of the Danysz, or mixed coliform, vaccine, preferably autogenous, usually cures them, while local ionization with zinc is of the greatest value.

EDITOR'S ABSTRACT.

LATE ETHER CONVULSIONS. R. F. WOOLMER and STEPHEN TAYLOR, Lancet **1**:1005 (May 2) 1936.

True convulsions occur only after full anesthesia has been established and must be differentiated from ether clonus, which occurs in the early stages of anesthesia.

The authors believe that ether anesthesia upsets the normal heat-regulating mechanism of the body, and they concur with Dickson Wright's opinion that heat stroke plays a big part.

By way of prophylaxis they caution against the preoperative use of atropine in excess of 1/150 grain (0.43 mg.) in children and against the use of excessive coverings.

LANGMANN, New York. [AM. J. DIS. CHILD.]

HISTOLOGIC PICTURE OF SURGICALLY CURED ABSCESS OF THE FRONTAL LOBE. B. KECHT, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141**:349, 1936.

Kecht shows that specimens in which an abscess of the brain completely cured by surgical treatment can be studied are rare, for most specimens examined at necropsy still show the signs of recent inflammation. For this reason he feels justified in reporting the case of a youth in whom an abscess of the frontal lobe was opened and cured, but who died five months later in an epileptic attack. Histologic examination of the region of the abscess revealed that the abscess had healed by connective tissue cicatrization and by glial proliferation, with degeneration of the neighboring medullary sheaths. Independent of this process of cicatrization there existed a fresh perifocal encephalitis, which the author regards as the cause of the convulsions and thus of the fatal outcome.

EDITOR'S ABSTRACT.

EFFECT OF BLOOD OF OTOSCLEROTIC PATIENTS ON VITALITY OF PLANTS AND ANIMALS. K. A. DRENNOWA, Monatschr. f. Ohrenh. **70**:826 (July) 1936.

Drennowa found in tests on flowers and tadpoles that the blood of patients with otosclerosis is less toxic than the blood of healthy persons. He thinks that this reduced toxicity of the blood of patients with otosclerosis is due to the lower choline content or to the higher epinephrine content. Since the dysfunction of the adrenal glands causes disturbances in the sympathetic nervous system, in the endocrine organs, in the intracellular metabolism, in the vasomotor system and in trophicity, the described experiments furnished an indirect corroboration of the hypothesis regarding the dependence of otosclerosis on the general trophic disturbances of the organism.

EDITOR'S ABSTRACT.

NEW APPARATUS FOR STUDY AND TREATMENT OF SPEECH AND VOICE. K. SPIELMANN and L. STEIN, Monatschr. f. Ohrenh. **70**:912 (Aug.) 1937.

Spielmann and Stein point out that experimental phonetics as well as logopedics and phoniatrics employs a number of registration apparatus but that most of them have defects. After mentioning some of the defects of laryngostroboscopes, they point out that the laryngostroboscope of Stein and Pollak-Rudin avoids disturbing and deceiving influences because it has no movable parts. They improved this model by changing the illumination, using a mercury vapor light spray.

They also describe an apparatus, the so-called photo-audion, which they constructed in order to be able to read the pitch and intensity of a sound on a scale. After describing the principle of this apparatus, they show that it is a valuable diagnostic and therapeutic aid. By means of this apparatus, the patient is able to watch his own voice formation on a scale. Moreover, it is helpful in teaching the sound language to deaf-mutes. Further, the authors describe a sound testing apparatus that is of value in the treatment of some patients afflicted with stammering and stuttering.

EDITOR'S ABSTRACT.

SEPTIC DISEASES OF TONSILLAR, ORAL AND NASAL ORIGIN. M. F. ZYTOWITSCH, Monatschr. f. Ohrenh. **70**:932 (Aug.) 1937.

Zytowitch summarizes the results of the investigations on this problem and reviews the researches that were conducted in his own clinic. He gives his attention to three problems: (1) whether there is a connection between the tonsils and the oral and nasal cavities, (2) whether there is a current of tissue plasm in the tonsils and what direction it takes and (3) whether the tonsils are organs of an endocrine character. In summarizing his observations he says that colloidal particles of dyestuff pass from the nasal and oral cavities into the tonsils. Under ordinary conditions these particles are not absorbed into the tonsils from the surface; so the author concludes that the tonsils cannot be regarded as the portal of entry for infection. He maintains that the nasal and oral cavities serve as portals of entry for infections. He observed that aqueous extracts of the tonsils influence the blood pressure and the vessels of surviving organs. Control tests with extracts from the muscles and mucous membranes exert such an action not at all or only slightly. Experiments on vagotomized rabbits seem to indicate that the tonsillar extract has a vagotropic action. The destruction of the tonsils changes the biochemical status of the organism, namely, the Dick and Schick reactions, the quantity of peroxidase and of blood sugar and the quality of the tonsillar extract. Tonsillectomy in young animals causes changes in the thyroid, adrenals and gonads. The author thinks that the tonsils belong to the endocrine organs. Clinical observations indicate that tonsillectomy frequently fails to counteract the symptoms for which it was performed. This is understandable in view of the fact that the tonsils do not present a portal of entry for infections. Because of the great physiologic significance of the tonsils, their removal is justified only in exceptional cases.

EDITOR'S ABSTRACT.

LOCALIZATION WITHIN THE CENTRAL VESTIBULAR SYSTEM. R. LEIDLER, Monatschr. f. Ohrenh. **70**:951 (Aug.) 1937.

In the preceding series of articles on the problem of localization within the central vestibular system, Leidler took up the inflammatory nonpurulent diseases of the brain stem. Now he discusses the problem of localization within the central vestibular system with regard to the vestibularly elicited positional changes of the head and the upper part of the trunk. It is known that in human subjects positional changes of the head or of the upper part of the trunk can be produced by experimental irritations of the vestibular system (by rotation and by galvanic irritation); however, these deviations are usually slight and generally stop after the stimulation ceases. The author reports the histories of patients in whom the

positional changes appeared chiefly after the stimulation ceased; they were usually of a considerable degree and persisted for longer periods. In estimating the observations on these patients in regard to the problem of localization within the central nervous system, the author emphasizes the following points: 1. Changes in the position of the head and of the upper part of the body as a reaction to vestibular irritation together with deviations of the eyes and with nystagmus indicate that the disease focus is within the spheres which are in the oral direction from the true central vestibular system, most likely in the oral direction from the anterior corpora quadrigemina at the level of the third ventricle. 2. Patients with such manifestations are less inclined to have vertigo of the vestibular type than are those in whom the disease process is more in the caudal direction, closer to the sphere of the vestibular system. 3. These positional changes develop generally only when vision is excluded and can usually be righted again by the patient as soon as the optical orientation is restored. Thus they are to a much greater extent dependent on superordinated mechanisms, as is the case in nystagmus, which is only quantitatively influenced by voluntary direction of the eyes but which cannot be entirely suppressed. On account of the great influence of the visual factor, the tests should be made only while the eyes are closed. 4. If vision is excluded, the patient fails to notice the positional changes, even if they reach severe degrees. 5. Many patients who show these positional changes react to vestibular stimulation with psychic changes, such as slight stupor, drowsiness or even loss of consciousness. 6. A process that is apparently entirely in the frontal or temporal portion of the brain may likewise cause such positional changes. In cases of such reactions it is likely that the vestibular center, which Spiegel assumes to be in the temporal portion of the brain, plays a part. On the other hand, it should be remembered that Gerstmann observed positional changes in cases of disease of the pole of the frontal brain and that Muskens was of the opinion that these changes are elicited by the basal ganglia. 7. An exact localization within the vestibular system is not possible as yet; however, it is probably certain that the positional changes described are not caused by the labyrinth, the vestibular nerve or its primary terminations in the brain stem.

EDITOR'S ABSTRACT.

HEMOSTASIS WITH PECTIN DERIVATIVE IN OTORHINOLARYNGOLOGY. J. BERBERICH, Monatschr. f. Ohrenh. **70**:966 (Aug.) 1937.

Berberich tried a new colloidal hemostatic, which contains pectin derivatives and which had been successfully employed in other branches of medicine, in the treatment of hemorrhages encountered in otorhinolaryngologic practice. In cases of nosebleed he applied cotton tampons that had been saturated with a 5 per cent solution of the hemostatic, and he found this treatment successful. He also used it in connection with operations, applying it either locally, to arrest the hemorrhage in the surgical field, or by intramuscular injection, to prevent postoperative hemorrhages. The preparation in his opinion is entirely harmless.

EDITOR'S ABSTRACT.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

IRA FRANK, M.D., *President*

Regular Monthly Meeting, Feb. 1, 1937

PULSION ESOPHAGEAL DIVERTICULUM. DR. H. G. LAREAU.

The patient was first seen on Oct. 27, 1936, when he complained of hoarseness, pain and difficulty in swallowing, with pressure in the neck after eating. There was a bulging sac on the left side of the neck at the level of the cricoid cartilage, which increased in size after eating. On pressure with the hand, food was emptied from the sac.

A roentgenogram (taken after a meal of barium sulfate) revealed a pushed-out pouch in the upper part of the esophagus. The anterior wall in the region of the epiglottis showed the configuration of a diverticulum. Fluoroscopic examination after a meal of barium sulfate revealed the same findings, and esophagoscopy verified the diagnosis of pulsion esophageal diverticulum.

Treatment consisted of a one step operation on Jan. 22, 1937. The end-results were good.

DISCUSSION

DR. JOSEPH C. BECK: A one step operation is not considered a good procedure in the majority of instances. The two step operation recommended by Lahey and performed by him many times is the one generally accepted. I have had the pleasure of seeing cure result in two cases in which this operation was used. The sac is turned upside down and emptied, and sutures are applied around it so that it is easily and permanently obliterated. With the one step operation, two complications may arise: mediastinitis, if there is extensive dissection, and dilatation at the ostium, which is always a danger.

DR. H. G. LAREAU: Dr. Beck is right in stating that the two step operation is the safest. However, two opinions are prevalent. One school favors the one step operation when the pedicle is small; in my case it measured about ½ inch (1.3 cm.). In such cases the one step operation is indicated. The other school favors a two step operation when the pedicle is large and the sac extends down into the thoracic cavity. With this operation, complications, such as mediastinitis, are minimized.

My patient was fed for four days postoperatively by a tube passed through the nose to the stomach, after which curds, egg nogs, etc., were fed by mouth. Recovery was uneventful.

TRANSPLANTATION OF FACIAL NERVE. DR. J. R. LINDSAY.

J. D., a man aged 47, gave a history of a simple operation on the mastoid on Oct. 26, 1934. Immediately after operation, the left side of the face was paralyzed. The patient was first seen in consultation on October 27. On October 29 I explored the mastoid cavity and found that the facial nerve had been torn out for a distance of about 1.5 cm. from the level of the horizontal canal downward. The canal for the facial nerve was also gone in this area. The accident apparently had occurred when the retrofacial cells were being cleaned out.

The upper end of the nerve was uncovered almost to the ganglion and the lower end to the foramen, and the ends were trimmed. A trough was prepared to receive the graft. A piece of intercostal nerve was removed from beneath the seventh rib and laid in the gap, with the ends in apposition with the trimmed ends of the

facial nerve. The graft was covered with silver foil. A graft of about 2.5 cm. was necessary to bridge the gap. Eight months later function began to appear at the angle of the mouth and gradually increased, until at nine months the muscles below and above the eye had regained function. The expression in repose is now good, and the patient is able to close his left eye. (Photographs and moving pictures taken before transplantation of the graft were presented.)

PITUITARY TUMOR EXTENDING INTO THE NASOPHARYNX: REPORT OF A CASE. DR. J. R. LINDSAY.

Mrs. F. C., aged 47, was seen by me in consultation on Dec. 30, 1936. The history of the illness was as follows: Nasal obstruction on the left side appeared sixteen months before and increased until both sides of the nose became almost completely obstructed. Deafness in the left ear was noticed fifteen months before, which persisted. A sudden decrease in the vision of the right eye was noted about three months before. Transitory occipital headaches, extending over the vertex, had occurred during the past three months.

Examination revealed a smooth mass filling the vault of the nasopharynx and extending down to the soft palate; the mass was also visible through the anterior nares. The left middle ear was filled with fluid (exudative catarrh). In the left anterior cervical region opposite the thyroid cartilage was a small soft nodule which possibly was metastatic. There was marked loss of vision in the right eye. The fundi as well as the external ocular movements were normal. General neurologic examination gave negative results. There were no signs of acromegaly and no symptoms of hypopituitarism.

Roentgenographic examination showed that the bone in the region of the sella turcica had been destroyed. There was gross destruction at the base of the skull. The clivus, petrous pyramids, pterygoid plates and base of the septum were extensively eroded.

Biopsy of a specimen from the mass in the nasopharynx revealed tumor tissue composed of a fairly homogeneous mass of cells, which when stained with hematoxylin and eosin showed up as having dark staining nuclei and diffuse faintly staining eosinophilic cytoplasm.

A diagnosis of probable chromophobic pituitary adenoma was made.

The patient received 700 mg. hours of radium therapy to the mass in the nasopharynx and is now receiving roentgenotherapy.

PITUITARY TUMOR INVADING THE NASAL SEPTUM: REPORT OF A CASE. DR. J. R. LINDSAY.

Three years ago I had another case in which almost the exact symptomatology and the same type of tumor were present as those described in the preceding case report. The patient was a man about 50 years of age, whose only complaint was that of increasing nasal obstruction of several months' duration.

Examination showed the posterior half of the septum to be dark red and so thickened as to almost obstruct the airway on both sides. The nasopharynx was clear. There was no visual disturbance. The visual fields as well as the ocular movements were normal. The patient exhibited no signs of disturbance of the pituitary gland.

Roentgenographic examination revealed practically the same degree of destruction of the pituitary fossa and base of the skull as in the first case.

Biopsy of a specimen from the mass invading the septum revealed tumor tissue which was almost identical with that in the first case.

A diagnosis of chromophobic pituitary adenoma was made.

The patient returned to his home city for radium and roentgen irradiation. Three weeks after treatment, he died rather suddenly from a cerebral accident, apparently hemorrhage.

(Lantern slides of roentgenograms and photomicrographs were shown to illustrate both case reports.)

The last two cases were presented because they represent a relatively infrequent type of tumor and because the symptomatology in both cases was referred entirely to the nose and ear in the earlier stages.

DISCUSSION

DR. SHERMAN L. SHAPIRO: In reference to Dr. Lindsay's first report, was the descending part of the facial nerve used?

DR. J. R. LINDSAY: Yes, the graft was taken from the posterior portion downward, close to the foramen.

DR. FRANK J. NOVAK: I have seen tumors much smaller than that shown by Dr. Lindsay in his second presentation, and yet the ocular symptoms were the first that were observed. With a large mass where the sella turcica formerly was, how does one account for the escape of the eye? How does it happen that the patient did not have pronounced ocular symptoms?

DR. J. R. LINDSAY: I do not think that I can answer Dr. Novak's question, except that one must infer that the tumor was not infiltrating. If it were, it would have involved other cranial nerves long before it grew so large. The tumor was very extensive and involved the base of the skull, yet it did not invade the cranial nerves.

INFECTIONS OF THE MAXILLARY SINUS. DR. CARL H. CHRISTOPH.

The case presented was one of a series of cases of chronic infection of the maxillary sinus in which a modified intranasal Canfield operation was done at the Illinois Eye and Ear Infirmary.

The technic of this operation is as follows: The incision is made anterior and at a right angle to the inferior turbinate, the entire mucosa of the lateral nasal wall being dissected from the bony nasal wall. A portion of the pyriform process as well as a large part of the lateral bony wall is removed, and a flap is laid into the antrum.

I believe that there is a great future for the intranasal operation. In my opinion as extensive an operation can be done through the nose as by way of the canine fossa. Halle recently made the statement that there is no indication for the Caldwell-Luc procedure except for a malignant condition; with the intranasal method, the bleeding is usually minimal, since the blood vessels are contained in the mucosa which has been dissected away from the bony wall, and one obtains an excellent view of the inside of the antrum. The diseased mucosa and polypi can be removed just as well as through a Caldwell-Luc incision, and I am certain that within a few years not many Caldwell-Luc operations will be performed.

DISCUSSION

DR. SHERMAN L. SHAPIRO: I should want to check the teeth for anesthesia after such an operation.

DR. WALTER H. THEOBALD: I wish to comment on the operation reported by Dr. Christoph, because eighteen or twenty years ago I wrote a thesis for candidacy for membership in the Chicago Laryngological and Otological Society in which I think I described practically the same procedure. By this operation the antrum was approached through the pyriform crest, and I reported its use on about fifty patients. I cannot say that I have repeated it as often since then. I have reverted more to the operation through the canine fossa. I titled the operation "a modified Canfield operation." If priority is being considered, I want to get my word in.

DR. JOSEPH C. BECK: I think that Dr. Christoph should wait until these patients have been observed for some time. Patients who are operated on in this manner often complain that the antral discharge empties without their knowledge while the head is bent forward. This is due to the fact that the most anterolateral part of the antrum at the floor of the nose is removed. They do not feel the secretion because there is so much disturbance to the nerve supply. That is

one of the great dangers from this operation, and Dr. Christoph may want to modify his remark that there will be few Caldwell-Luc operations performed in the future.

DR. SAMUEL SALINGER: I can remember Dr. Theobald's presentation of this operation many years ago and recall that it was well received. I have done the operation successfully a number of times, although in one case I drilled for a long time before being able to enter the antrum. There are patients whose antral walls are very thick due to the small size of the antrum, so that in such a case the pyriform margin represents a double thickness which is difficult to penetrate. I should consider the operation only when the roentgenogram reveals a large antrum with comparatively thin bony walls.

DR. CARL H. CHRISTOPH: I thought that I should be sat on. I have not been using this operation as long as Dr. Theobald, and I may not find it to be as efficient as I now think it is. The advantages as I see them are: First, one does not see the patient the day after the operation with his face badly swollen; second, the patient does not complain of anesthesia of the teeth and, third, there is little reaction after the operation. I do not suppose that it can be used in all cases. Perhaps, as stated by Dr. Salinger, the small antrum with the thick wall is not a favorable field. I think that the reason for failure in most cases is that the procedure is not radical enough. The lateral wall is dissected as far back as the posterior end of the inferior turbinate. No ridge is left on the floor of the nose, and the procedure is such an extensive one that closure does not occur. In answer to Dr. Beck as to the secretion running from the nose when the patient stoops over, none of the patients has complained of this up to the present, but perhaps because a long enough time has not elapsed since operation.

LYMPHOSARCOMA OF THE NASOPHARYNX AND NOSE: REPORT OF A CASE. DR. WILLIAM A. SMILEY.

Mr. J. M., aged 47, came to my office on Aug. 4, 1936, with the following history: For the past four months he had been troubled with considerable bloody discharge from the left nostril and an increasing nasal obstruction. He had consulted a physician, who had on three occasions removed "polyps" from the left nostril. About six weeks before the present examination, the right side of the nose also became obstructed. Three weeks before the patient noticed a swelling in the right side of the neck, which increased rapidly in size. It was not tender, nor was there any nasal pain or headache. No loss of appetite, weight or strength had occurred.

Examination revealed complete obstruction of each side of the nose by a tumor mass, which on the left side protruded from the anterior nares and also from the posterior nares, where it was continuous with the vault of the pharynx. The tumor was rather firm and bled easily. There was a cervical lymph node about 8 cm. in diameter on the right side. Roentgenograms of the nasal sinuses showed marked opacity of the left antrum and partial opacity of the right. A roentgenogram of the chest showed nothing abnormal. The differential blood count and the Kahn and Wassermann reactions were negative.

A portion of the nasal tumor mass was removed and sent to the laboratory. There was little bleeding. The diagnostic report was as follows: "Round cell lymphosarcoma. The fact that most cellular tumors in this region are transitional cell epidermoid cancer was duly considered in making the diagnosis." The cervical gland was not disturbed.

The patient was referred to Dr. Cutler, at the Michael Reese Hospital. He concurred in the diagnosis and started radiation therapy. Using the 4 Gm. pack, he gave a total of 162,000 mg. hours to the nose and neck over a period of seven weeks. At the end of that time the tumor mass in the nose had completely disappeared, and the cervical gland was barely palpable. Nasopharyngeal examination gave negative results. There was a large perforation in the posterior part of the nasal septum.

In November the patient was given 50 mg. hours of intranasal radiation on each side. At present he is free from nasal symptoms except dryness and is in active supervision of his business. He complains of some gastric distress, which may be a forerunner of a distant involvement.

Kundrat, of Vienna, in 1893 was the first to describe lymphosarcoma adequately (Ueber Lympho-Sarkomatosis, *Wien. klin. Wchnschr.* **6**:211, 1893). According to him, it is a tumor originating in a single lymph node or a group of nodes, as in the pharynx, tonsil or intestinal tract. It consists of a reticulum in which there are large numbers of lymphocytes, and after destroying the capsule of the node it extends to other nodes.

Ewing, in a series of 100 cases of nasopharyngeal malignant conditions, found 9 instances of lymphosarcoma (Radiosensitivity, *Radiology* **13**:313 [Oct.] 1929).

The site of origin of nasopharyngeal lymphosarcoma is usually the lymphoid tissue of the nasopharynx. The tendency of the growth is to extend forward, involving the nasal sinuses, and in a case reported by Cutler it even caused complete blindness (Lymphosarcoma, *Arch. Surg.* **30**:405 [March] 1935).

In making a differential diagnosis, one must consider in addition to squamous cell carcinoma, transitional cell carcinoma, lympho-epithelioma and aleukemic leukemia.

The clinical characteristics of this tumor are its marked radiosensitivity and its equally marked tendency to local and outlying recurrences. These in turn respond to radiotherapy, but repeated recurrences are the rule, and the ultimate prognosis is bad.

DISCUSSION

DR. FRANK J. NOVAK: It might be interesting to know what the original polyps were that were removed from the nose—whether they were typical nasal polyps, which most of the otolaryngologists scorn to examine microscopically, or whether they were true neoplasms. Were those original polyps sectioned and examined?

DR. WILLIAM A. SMILEY: No, they were not. The patient consulted a general practitioner who was a personal friend of his, and he consented to remove the polyps.

The reason the patient is not here is that he has been having some gastric distress, and it seems to me that there is a possibility that some distant metastases may be developing.

PRIMARY CHOLESTEATOMA OF THE MASTOID. DR. L. B. BERNHEIMER.

The purpose of this communication is (1) to present a case of cholesteatoma of the mastoid without involvement of the middle ear and (2) to discuss briefly the etiologic mechanisms which may produce this type of disease.

W. R., a man aged 29, presented himself for examination because of temporal headache on the right side and a discharge from the right ear which had persisted for six weeks. He had had no earache, tinnitus, apparent reduction of hearing or infection of the upper respiratory tract. There was no history of previous aural trouble, except that twenty-four years prior to the present examination a foreign body had been removed from the right auditory canal through a Wilde incision.

Examination revealed a postauricular scar on the right side. Both drums were mildly retracted but were otherwise normal. The right auditory canal contained an amount of serous discharge which came from a fistula surrounded by a small amount of granulation tissue in the posterior wall of the canal. These granulations were infected and proved to be the sole source of the secretion seen in the canal.

Antrotomy was performed on the right side, during which the following findings were noted: The cortex was intact. The bony wall of the canal showed no evidence of either surgical or infectious destruction, except for a small perforation through its posterior aspect. Removal of the cortex exposed a large cholesteatoma which had destroyed most of the cellular structure of the mastoid and a great part

of the dural plate over the posterior and middle fossae. An excellent view of the antrum was obtained. It was found to contain no discharge and was otherwise normal. The cholesteatoma itself was clean and odorless, having the gross appearance of a large pearl. Microscopically, large nests of cholesterol crystals were seen, through which endothelial lymphocytes and giant and epithelial cells were distributed. No granulation tissue or pus was encountered throughout the entire mastoid.

Cholesteatoma of the mastoid may result from chronic infection, primary congenital growth or a displacement of epithelial cells into the mastoid through growth, trauma or a combination of both. In the case just described no evidence of aural infection, either acute or chronic, was encountered, with the exception of the infected granulation tissue found in the external auditory canal. However, it is unlikely that either the fistula or the granulations had been present for more than the six week period of the aural discharge, so that it is illogical to conclude that the cholesteatoma was secondary to an infectious process in the canal.

The possibility that epithelial tissue was displaced into the mastoid at the time of the removal of the foreign body must be considered. Unfortunately, the otologist who performed the operation is deceased, and no hospital records could be found. However, at the time of antrotomy the cortex and bony canal were intact, not even evidence of regeneration of bone being encountered. In the light of these findings, it is unlikely that removal of the foreign body included any operative procedure on the bone, so that it is difficult to explain how epithelial tissue could have been displaced into the mastoid at this time.

Since it is unlikely that this cholesteatoma resulted from either chronic infection or displaced tissue, primary congenital growth must be considered.

Teed's monograph on cholesteatoma verum tympani was then reviewed.

DISCUSSION

DR. ALFRED LEWY: Dr. Bernheimer's patient was also seen by me, and my findings were essentially the same as those described by him. The cavitation in the mastoid shown in the roentgenogram was interpreted by me as a surgical cavity from the previous operation or a result of that. The patient had had a rubber eraser deep in the canal, and some operative work had been done on the bone when the eraser was removed. I think that the evidence is not clear that the present condition was a primary cholesteatoma associated with a suppurative lesion which may have existed longer than the patient believed. I recently saw a cholesteatoma that had existed for many years. The young man, a musician, had undergone an operation on the mastoid several years before I saw him. He had had pain in the region of the mastoid. Two years ago he went to a large clinic and was sent home without any surgical work being done. After his money was all gone, he came to the Illinois Eye and Ear Infirmary. The mastoid was inflamed, but the canal was dry and there was partial atresia. I had him examined by a neurologist, who made a diagnosis of hysteria. I was not satisfied with the diagnosis and kept him under observation. Within two weeks acute otitis media developed with discharge. The discharge had not been present for some time before that. The patient was treated and kept under observation. The scar became red, but there was no fluctuation. Because of the general appearance of the patient and the continued and increasing symptoms, operation was decided on. Evidence of a former incomplete operation was encountered, because the operator discovered the sinus lying in front of the antrum, and the operation had to be completed by the Küster procedure. The antrum was very deep. (A roentgenogram was presented.) This (indicating on the roentgenogram) was interpreted as the operative cavity, which was not there. The cavity was filled with a large cholesteatoma. The operation was performed only three days ago, and the patient is doing very well.

In summary, a cholesteatoma due to a suppurating process produced no symptoms except pain (in a neurotic patient) over a period of years. Now the pain is gone, and the patient feels all right.

DR. GEORGE E. SHAMBAUGH JR.: Did Dr. Bernheimer merely remove the cholesteatomatous material or did he do a radical operation on the mastoid?

DR. SAMUEL SALINGER: It is generally agreed that a cholesteatoma that shows any connection with the tympanic cavity or mastoid antrum cannot be considered a true or congenital tumor. There must be a definite partition of bone separating the mass from any part of the tympanic air spaces. Most of the true cholesteatomas are found outside of the dura mater near the squamoparietal or the parietooccipital junctures and occasionally close to the dura in the petrosal bone itself. In the literature which I have read during the past twenty years, I can recall only two genuine cases of primary cholesteatoma of the temporal bone on record. One case was that of a man who was brought into the hospital with acute mastoiditis and was operated on at once without the usual preoperative roentgenogram being made. A radical operation was performed, and two weeks later a picture was taken; to every one's astonishment there was a smooth cavity posteriorly that seemed to be overlying the mastoid. A second operation disclosed a true cholesteatoma which was separated from the cavity by a dense lamella of bone.

In the second case the cholesteatoma was in the petrosal bone and was demonstrated roentgenographically. Spinal fluid was withdrawn, and a radical operation was performed on the mastoid, the middle fossa and superior surface of the petrosal bone being exposed. At this point in the procedure the operator encountered a cavity just beyond the labyrinth, a cavity from which large amounts of cholesteatomatous material and part of the matrix were removed. While in Dr. Bernheimer's case there is no definite history of infection, nevertheless the history of trauma and the involvement of the periantral region would lead one to believe that the resultant tumor formation was more than likely due to secondary invasion of epithelial tissue rather than to congenital inclusion.

It has been shown by Witmaak and others that extreme retraction of Shrapnell's membrane may cause an invagination into the attic and antrum leading to the formation of cholesteatoma in spite of definite absence of history of any previous suppuration.

DR. WALTER H. THEOBALD: I believe that the question of differentiating between primary and secondary cholesteatoma is debatable, but the younger the child, the more likely one is to find primary cholesteatoma. By a peculiar combination of circumstances, I saw a case last week. The patient is now about 26 years old and was first seen by Dr. Pierce in October 1914. He was then about 3 years old and had had a discharge from the ear for three months without any known cause. He had no pain or other symptoms referable to otitis media. He had been under the care of pediatricians, and suddenly a creamy discharge was noted coming from his ear. Polypi were removed, and subsequently a radical operation on the mastoid was advised. At the time of operation a diagnosis of primary cholesteatoma was made. Cholesteatoma was found invading the mastoid, although there had been no evidence of the cholesteatomatous material invading the middle ear. A dry ear was obtained subsequently, after an extremely trying time. The child required many months of after-treatment. The hearing was totally absent then and has been ever since. When I saw the patient a week ago the ear was again moist, but there was no evidence of cholesteatoma. This is one of those unfortunate cases in which moisture in the ear recurs.

DR. L. B. BERNHEIMER: In answer to Dr. Shambaugh, simple antrotomy was done. The antral lining was found to be clean. As the drum membrane was normal, no indication for a radical operation was seen.

The one point that speaks against primary cholesteatoma is that a postauricular incision had been made to remove a foreign body. It is possible that epithelial tissue was displaced into the mastoid at that time. However, as no evidences of operative work having been done on the bone were encountered at the time of antrotomy, it is difficult to understand how much displacement could have taken place.

The cholesteatoma followed Dr. Salinger's rule for primary cholesteatoma, as it was walled off from both the antrum and the middle ear by trabeculae of normal bone.

As there was no history of a previous otitis and as no evidences of infection were encountered in the mastoid, the cholesteatoma in this particular case was in no way parallel to the cholesteatoma described by Dr. Lewy.

THE HUMAN TOOTH AS A RECORDER OF BIOLOGIC PROCESSES WITH SPECIAL REFERENCE TO THE NEONATAL RING. DR. ISAAC SCHOUR.

Experimental and clinical studies have established the fact that the growing tooth is a delicate and accurate indicator of biologic processes. Erdheim's analogy that the dentin of the incisor of the rat acts like the drum of a kymograph in its response to disturbances in calcium metabolism is applicable to enamel as well as to dentin, to various metabolic changes and to the growing human teeth.

Injections of sodium fluoride or alizarin produce a permanent mark or ring in the portion of the tooth that is calcifying at the time of the injections. This method makes possible the accurate measurements of the rate of growth of enamel and dentin. In the incisor of the rat there is a 16 micron daily incremental rhythm. In the human tooth the rate of growth is approximately one-fourth as fast.

In the attempt to analyze the rings that were produced experimentally, a number of other rings have been discovered, each of which is constant and characteristic in position and is therefore produced by a common cause. One of these constant rings is the neonatal ring which is found in every human deciduous tooth and which constitutes a permanent biologic landmark. The neonatal ring mirrors the biologic changes in the dental tissues of the new-born child that may result from metabolic influences that are incident to a change from intra-uterine to extra-uterine existence.

The presence of other constant physiologic rings as well as the occurrence of rings induced by known diseases indicate the value of an analysis of rings in teeth as an aid in the diagnosis of health and disease.

DISCUSSION

DR. WALTER H. THEOBALD: This has been a very dramatic and fantastic story. Dr. Kronfeld, of the Dental College of Loyola University, has been much interested in this work, and I have invited him to discuss Dr. Schour's presentation as well as to provide a link between this work and the sinuses. Dr. Kronfeld has made an exhaustive study on the maxillary sinuses in relation to the teeth.

DR. RUDOLPH KRONFELD: I certainly appreciate Dr. Theobald's invitation to hear Dr. Schour's report. I have known of his investigations for many years and have followed them with great attention and appreciation, for I have felt, as he pointed out, that science, especially morphology, has reached a point where it is no longer sufficient simply to say that a structure or tissue grows, but one wants to know how much it grows in a certain length of time and then proceed to an understanding of the anomalies in pathologic events. I hope that every one enjoyed Dr. Schour's presentation as much as I did.

Dr. Theobald asked me to present some material on the pathologic structure of the teeth and the maxillary sinus. My work has been along a little different line. What I have done is to examine about eighty jaws from persons ranging in age from birth to 80 years. All were studied roentgenographically and then decalcified and studied in sections. From this material, I can tell, for instance, how far the teeth are developed in the jaws of a child of 1, 2, 3 and 4 years of age. I can study the normal and abnormal tissues of the jaw; in the latter I may find many cysts and other pathologic conditions, and I can then compare what is found microscopically with the clinical picture.

In sectioning the upper jaws I have always paid attention to the maxillary sinuses and their relation to the root ends in normal and abnormal conditions as well as the relation of the nose to the incisor teeth. I shall show by means of lantern slides a few things which I believe will be of interest.

(Lantern slides were presented showing the growth of the maxillary sinus in children, the relationship between the root ends of the upper posterior teeth and the floor of the antrum and the effect on the antrum of granuloma and cysts originating from the upper teeth.)

DR. WALTER H. THEOBALD: I should like to ask Dr. Schour regarding the failure of enamel to develop, such as one sometimes sees in the central incisors.

DR. ALFRED LEWY: I should like to know what dilution of sodium fluoride was used.

DR. H. G. LAREAU: With reference to the line markings on teeth as described by Dr. Schour, I should like to ask him if he could deduce the possibility of these markings being used in a practical sense by horse buyers and traders, who are able to tell the age of a horse within a few months.

DR. I. S. SCHOUR: I want to state that these illustrations and models which have been shown were made principally by Dr. Spence, who assisted me in presenting this material.

In regard to the question of hypoplasia of the enamel, no doubt many have noted definite pits on the central incisors and the cuspids but usually not on the lateral incisors. These are disturbances that occur when the enamel is calcified. By checking up on the amount of enamel and dentin on a tooth, we can tell definitely when these marks are formed in the early years of the life. We have further evidence in that the central incisors and cuspids will show this defect but not the lateral incisors. Dr. Kronfeld has found that the lateral incisors start forming much later than the central incisors and cuspids, usually at about the second year. By this we know that these changes occur before that time. The probability is that these pits are found in the teeth of persons who had tetanic convulsions at about that time. Convulsions are more likely to occur about the last part of the first year. Sometimes there will be a history of convulsions during that time and also during the third year, but no lines form in the enamel during the latter period. The explanation is that the first year of life probably marks the most rapid period of growth in the life of the child. When the child grows rapidly, the disturbances of calcium metabolism are more effective. When there is slower growth, these changes may not be registered.

In answer to Dr. Lewy, we used about a 2 per cent solution of sodium fluoride, and 1 or 1.5 cc. was sufficient. The injections were given intravenously, which made it simple. There were no markings of any sort.

With relation to marks on teeth, it is quite evident that if the neonatal life is sufficient to produce marks there must be other experiences that are recorded in the teeth. We are now studying changes in rats and are finding weaning rings. When we put animals on one diet and then change to another, we find differences recorded in the teeth. When the mother stops giving the child milk there is a change in diet, and this sudden change produces a definite ring in the teeth. This ring not only acts as a boundary but also indicates changes in calcification. It is logical to expect to find a ring indicative of puberty. We are almost confident that we shall find such a mark, but we shall have to look for it in the third molar. It is possible that we shall be able to tell whether a third molar comes from a female or a male, for puberty occurs earlier in the female.

With relation to telling the age of the horse by examining the teeth, my impression is that this depends not on the rings but on the degree of abrasion which progresses with age.

DR. RUDOLPH KRONFELD: There is a difference in the structure of the teeth of man and of the horse. In man there is only one layer of enamel, whereas in the horse the enamel is folded in, so that there is a funnel-shaped layer of enamel inside the tooth. As the animal grows older, the outer layer of enamel stays about the same width, but the inner layer grows narrower and narrower as the teeth wear down. Thus, from the difference in the size of these two enamel rings on the abraded surface it is possible to tell the age of a horse.

DR. I. S. SCHOUR: An instance in which the age of an animal could be told happened in Switzerland. A man was arrested for hunting a stag, with the accusation of breaking the law, for it is against the law to hunt a stag that is less than 6 months of age. This man brought a dentist for a witness, who by counting the number of rings in the teeth was able to tell the exact age of the stag, because the annual ring is formed in the secondary dentin.

AMERICAN OTOLOGICAL SOCIETY

EDMUND PRINCE FOWLER, M.D., *President, Presiding*

Seventieth Annual Meeting, Long Beach, N. Y., May 27-28, 1937

ABSTRACTED BY DR. ALFRED LEWY, CHICAGO, FOR DR. ROBERT SONNENSCHEIN

(Concluded from December [page 792])

Friday Afternoon Session, May 28

REPORT OF NEW YORK COMMITTEE ON OTITIC MENINGITIS. DR. JAMES G. DWYER, New York.

The New York committee on meningitis has continued its work during the last year, and in making our annual report we point out that for the first time we are able to report progress in several directions, which will be elaborated on further.

During the last year the literature everywhere has been full of reports covering cases of otitic meningitis, with a larger percentage of recoveries than ever before. These reports have been complete, and the bacteriologic work has been exact. This is a distinct step forward, as the bacteriologic diagnosis in the past has been confusing and unsatisfactory. The use of sulfanilamide in its various forms and the combined use of this drug with allied substances has been a new development, which will be gone into later in this report in some detail.

The committee received reports with complete records covering 42 cases. In addition, over 30 other cases were reported indirectly to us. Persistent cloudiness of the spinal fluid, without organisms, means that the infection is still extradural and is probably due to a sequestrum or a localized collection of pus; recognition of this fact has saved the lives of several patients in this series.

Sulfanilamide and Its Related Compounds.—The use of the various forms of sulfanilamide and its related compounds has given the greatest hope physicians have had in years in the treatment of meningitis due to Streptococcus haemolyticus. The reports from all over the country are encouraging, but a note of warning should be injected at this stage of a new therapy, as a swing of the pendulum may go too far one way, which has been the history with most new drugs.

Method of Action of Sulfanilamide: Dr. F. P. Gay, professor of bacteriology at the Columbia University College of Physicians and Surgeons, has found that the drug acts through stimulation of the mononuclear leukocytes. He has given permission to report these findings on the action of sulfanilamide.

Method of Use: One can administer the several preparations of sulfanilamide alone by mouth or can inject intramuscularly one of the complex derivatives (known as prontosil) in which the simple form is combined with a dye radical. One can use ordinary sulfanilamide by mouth combined with the crystalline form by the subcutaneous, intramuscular or spinal route. Prontosil is not given intravenously or intraspinally. The crystalline form of sulfanilamide is dissolved in an 0.8 per cent saline solution. In some desperate cases ordinary sulfanilamide has been given orally, prontosil intramuscularly and a solution of crystalline sulfanilamide intraspinally.

Dr. Josephine Neal, of the board of health of the city of New York, reported on a series of 11 patients with meningitis due to Str. haemolyticus treated with sulfanilamide and prontosil (the disodium salt of 4-sulfamidophenyl-2'-azo-7'-acetylamino-1'-hydroxynaphthalene-3',6'-disulfonic acid). There were nine recoveries. Dr. Daniel Cunning, of the Queensboro Hospital and the Manhattan Eye, Ear and Throat Hospital, reported a series of 9 cases with 6 recoveries. The cases are not included in our series. These results are startling when one considers the reports of previous years.

Dr. Neal recommends the use of from 5 to 10 cc. of prontosil every four hours, the dose depending on the size of the patient, until the temperature has declined.

At the same time from one to three of the 5 grain (0.32 Gm.) tablets of sulfanilamide should be given every six hours. The oral administration should be continued for at least a week to ten days after the temperature has become normal. Dr. Cunning opened the dura in some cases, put the patient in the Trendelenburg position and used the drug freely. In some cases he gave a solution of the crystalline form of sulfanilamide intraspinally.

The future looks a little brighter for the solution of this problem, but as physicians have followed the rainbow so long they naturally have to go slowly before they come to any conclusion. I make this report for the New York committee as chairman and wish to acknowledge the hearty cooperation of all the members.

REPORT OF THE CHICAGO COMMITTEE ON OTITIC MENINGITIS. PART I. DR. ALFRED LEVY, Chicago.

This is an abstract of the report, being a series of recommendations based on the investigations and experience of the committee.

When a patient with meningeal symptoms is observed, search is made for an aural or a rhinolaryngeal source, and a general neurologic examination, including examination of the spinal fluid, is done. If a discharging ear is present, without waiting for the result of cultures of the spinal fluid and unless a positive diagnosis of a nonsurgical type of meningitis can be immediately and positively established, one should proceed at once to mastoidectomy and uncovering of the dura of the middle and the posterior fossa over an area of about 2 sq. cm. Further surgical treatment of the petrous apex and the labyrinth and drainage of the cisterna pontis lateralis are recommended, according to indications in the particular case.

In support of this view, 3 cases are reported. All the patients entered the hospital with a diagnosis of meningitis.

1. The first patient had 11,000 cells in the spinal fluid, and the condition was diagnosed as a lesion of the posterior fossa. A large epidural abscess and pachymeningitis were uncovered; temporary improvement was noted, with a subsequent increase in intracranial pressure. Drainage of the cerebellar abscess was followed by recovery.

2. The second patient had chronic suppurative otitis media, a stiff neck, facial paralysis, vomiting, a temperature of 103 F. and 3,100 cells in the spinal fluid. Sinus thrombosis was found at operation; drainage of the sinus and ligature of the jugular vein were followed by recovery except from the facial paralysis.

3. The third patient entered the hospital with a condition diagnosed as epidemic meningitis. Otitis media was discovered; the spinal fluid showed 675 cells. Mastoidectomy and uncovering of the dura were followed by recovery.

For the present, hope lies in recognition of the proper time for preventive operation and of its scope, whether simple, radical, into the petrosa or into the labyrinth. Too early surgical intervention in uncomplicated mastoiditis is productive of mischief and does not prevent complications.

As a procedure in cases of mastoiditis of violent onset, which sometimes leads to fulminating meningitis, I can only recommend watching the cell count of the spinal fluid, as recommended by Lund for labyrinthitis, and operating at the first evidence of meningeal involvement. The patients with or without chills and the temperature curve characteristic of venous sepsis from the beginning often have bacteremia with incidental otitis and should have the benefit of blood transfusions or convalescent serum before the sinus is exposed.

The sulfonate dyes may be of use in treating these conditions. I have personally observed 2 authentic cases of otitic meningitis due to a hemolytic streptococcus with recovery and know of 4 others, all recent.

Directory of Otolaryngologic Societies *

FOREIGN

COLLEGIUM OTO-RHINO-LARYNGOLOGICUM AMICITIÆ SACRUM

President: Prof. H. Burger, Keizersgracht 317 C, Amsterdam.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: San Francisco. Time: June 13-17, 1938.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

AMERICAN BRONCHOSCOPIC SOCIETY

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Place: Atlantic City, N. J.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. J. F. Barnhill, Miami Beach, Fla.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Place: Atlantic City, N. J.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Samuel J. Kopetzky, 71 E. 80th St., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Atlantic City, N. J. Time: April 27-29, 1938.

SECTIONS:

Eastern.—Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Philadelphia. Time: Jan. 7, 1938.
Southern.—Chairman: Dr. Murdock S. Equen, Medical Arts Bldg., Atlanta, Ga.
Place: Atlanta, Ga. Time: Jan. 24, 1938.
Middle.—Chairman: Dr. James B. Costen, Beaumont Bldg., St. Louis.
Place: St. Louis, Mo. Time: Jan. 26, 1938.
Western.—Chairman: Dr. Arthur C. Jones, 105 N. 8th St., Boise, Idaho.
Place: Santa Barbara, Calif. Time: Jan. 30-31, 1938.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Atlantic City, N. J.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.